A STUDY ON THE ROLE OF PERIOPERATIVE PARATHORMONE LEVEL AFTER TOTAL THYROIDECTOMY AS A PREDICTOR OF HYPOCALCEMIA

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CERTIFICATE

This is to certify that the dissertation titled **"THE ROLE OF PERIOPERATIVE PARATHORMONE AFTER TOTAL THYROIDECTOMY AS A PREDICTOR OF HYPOCALCEMIA"** is the bonafide original work of **DR.S.SUGANESWARAN** in partial fulfillment of requirements for **M.S. BRANCH I (GENERALSURGERY)** Examination of the Tamilnadu DR. M.G.R. Medical University to be held in APRIL 2014. The Period of study was from May 2013 to December 2013.

Prof. D. NAGARAJAN M.S.
Professor
Department of General Surgery,
Government Kilpauk Medical College and Hospital,
Chennai.

Prof.P.N.SHANMUGASUNDARAM M.S.
Professor and Head
Department of General Surgery,
Government Kilpauk Medical College and Hospital,
Chennai.

PROF.P.RAMAKRISHNAN M.D. D.L.O

DEAN

Government Kilpauk Medical College and Hospital,

Chennai-10

ABSTARCT

INTRODUCTION:

Thyroidectomy is the most common endocrine surgery performed. Hypocalcemia is one of the serious complication following Thyroidectomy, incidence varies from 20-30% occurs due to devascularisation or accidental removal of parathyroid gland. To manage postoperative hypocalcemia, most practitioners obtain serial serum calcium monitoringand respond appropriately to low levels. Recently, several studies have advocated measurement of the parathyroid hormone (PTH) level several hours after operation in order to predict the development of hypocalcemia.

AIM:

In this study we assess the role of Parathormone level after Total thyroidectomy in predicting the complication of hypocalcemia and to correlate the serum Parathormone and corrected calcium level after Thyroidectomy.

METHODS:

48 patients admitted and underwent total thyroidectomy from January 2013 to December 2013 were included in this study. This study was approved by the ethical committee and informed consent was obtained from all participating patients. Serum calcium were measured before surgery. Postoperative parathormone and calcium were done and correlated following surgery.

RESULTS:

Total thyroidectomy was the procedure done in all 48 patients. 22 out of 48 patients (45.8%) mostly in the middle aged group developed postoperative hypocalcaemia , 18 patients (37.5%) had preoperative serum calcium less than 9.0g/dl . Low preoperative serum calcium significantly correlate with fall in post operative serum calcium. 16 patients had decreased post operative Intact PTH and

all of them developed hypocalcemia suggesting a positive correlation between serum PTH and serum calcium done post operatively.

CONCLUSION:

Hypocalcemia following total thyroidectomy remains a significant cause of morbidity and increases the cost of procedure due to prolonged hospital stay. Use of serum PTH postoperatively 6hrs following total thyroidectomy significantly predict the complication of hypocalcemia. Proper identification of the parathyroid gland and preserving the gland with adequate vascularity significantly reduce the complication.

KEYWORDS:

Post Thyroidectomy Hypoparathyroidism, Intact Parathormone, Hyocalcemia.

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INTRODUCTION

Thyroidectomy is the common endocrine surgery performed worldwide.

After Diabetes and Obesity , thyroid disorders are the most common cause of metabolic disturbances and surgery is the mainstay of treatment.

Total Thyroidectomy is accepted as the standard surgical procedure for the management of Multinodular Goitre and Thyroid malignancies.

Thyroid gland has complicated anatomy , and its relation to vital structures of the neck pose a great challenge for the surgeon. Thyroid surgery in experienced surgeons is one of the safest procedures performed.

Complications like hematoma and seroma can be easily managed,

but recurrent laryngeal nerve injury and hypocalcemia are quite significant and life threatening complications. Hypocalcemia is the most common post surgical complication secondary to Hypoparathyroidism, incidence ranging from 20- 30%. Post Thyroidectomy Hypoparathyroidism is usually due to disruption in vascularity to the parathyroid gland, hence the surgeon should be meticulous in the preservation of parathyroid gland with adequate vascularity.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

THYROID GLAND ANATOMY

Thyroid gland has two lobes, isthmus and a pyramidal lobe. It extends from C5 to T1vertebra. In adult , the normal thyroid gland weighs about 30 g in the adult. Each lobe measures 5 cm in length, 3 cm in width, and 2-3 cm in thickness. The isthmus connecting the two lobes is about 1.3 cm in breadth.[1]

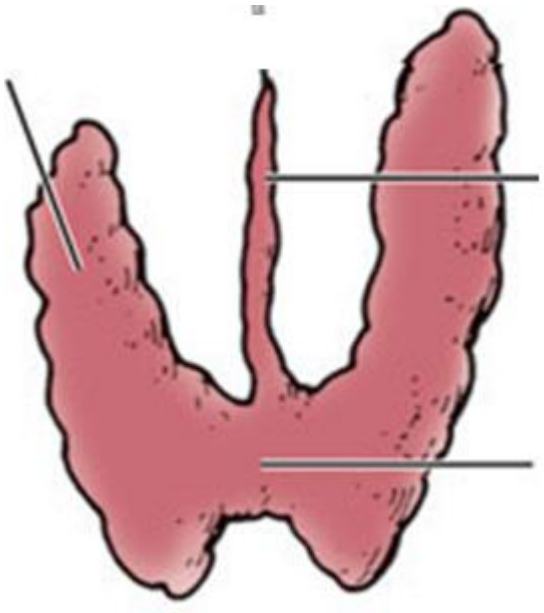


Figure 1; THYROID GLAND

VASCULAR SUPPLY

Thyroid gland has the extensive blood supply competing with the adrenals .[2]

Two paired arteries, the superior and inferior thyroid arteries, and an unpaired midline artery , thyroid ima, supply the thyroid and parathyroid gland.

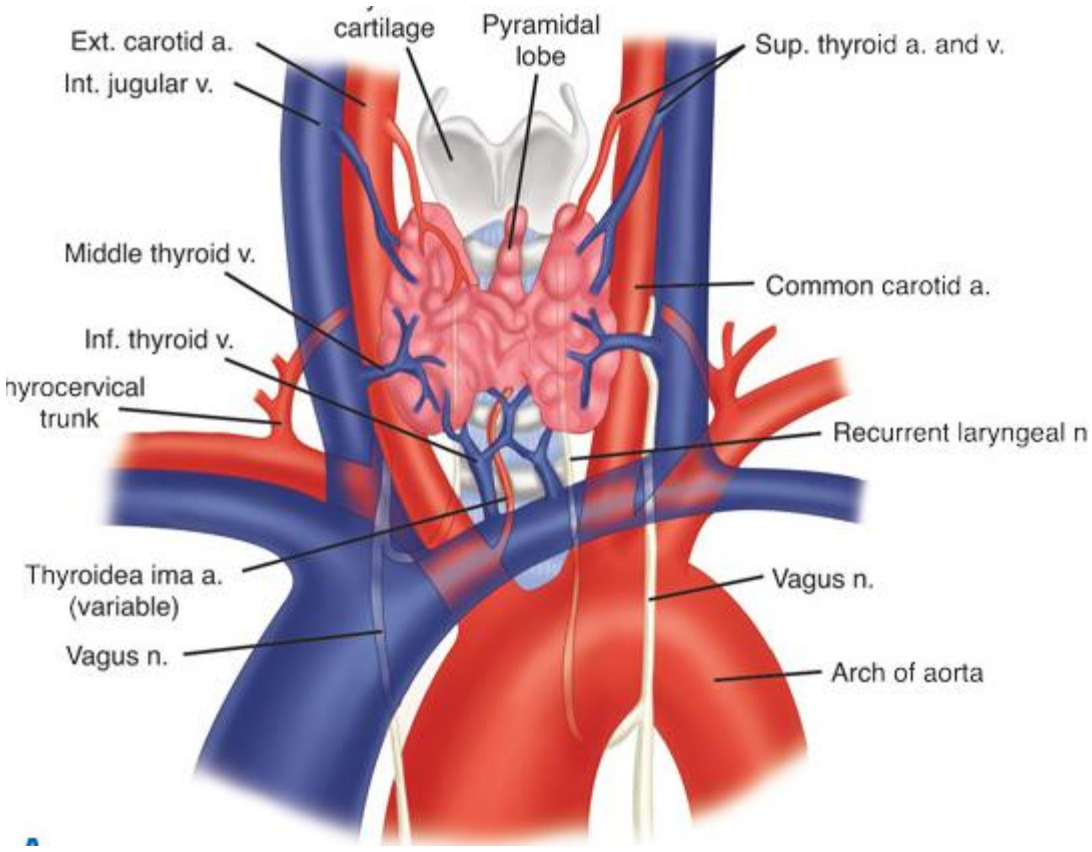


Figure 2: BLOOD SUPPLY OF THYROID GLAND

ARTERIES

The **superior thyroid artery** is a branch of external carotid artery. It arises just above or below or at the bifurcation of the common carotid artery. At the superior pole of thyroid gland, it divides into anterior and posterior terminal branches supplying most of the thyroid gland .

The posterior branch, supplies the superior parathyroid gland .[3]

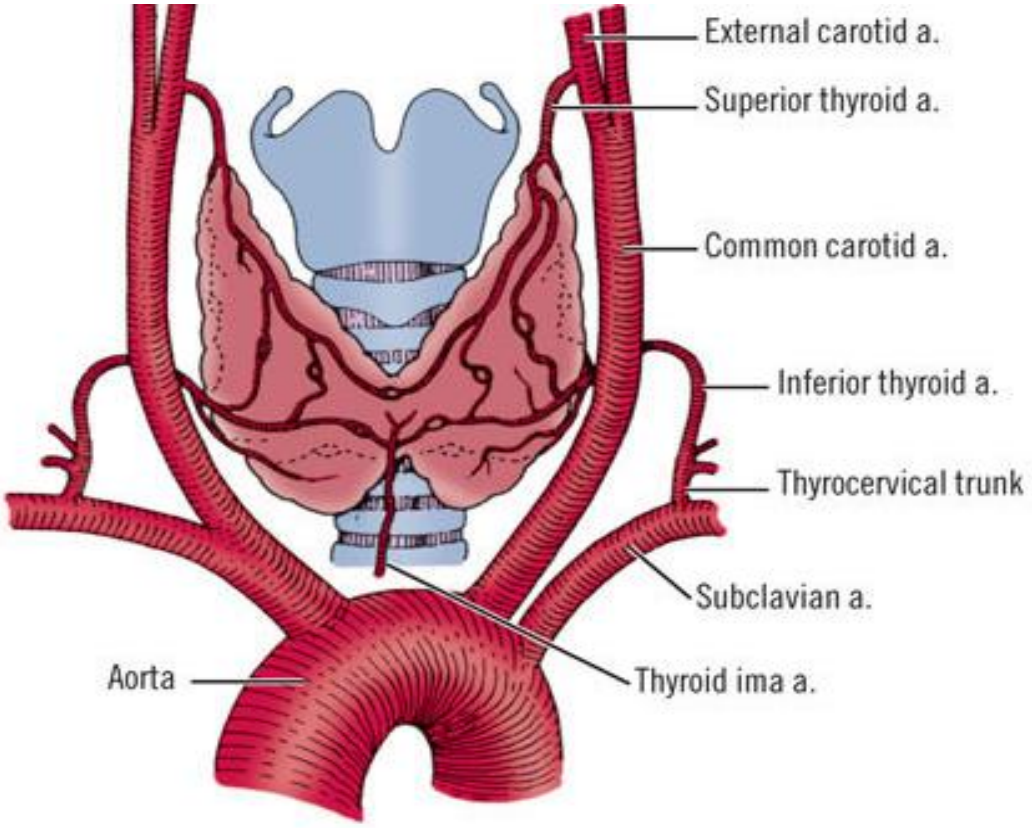


Figure 3: ARTERIAL SUPPLY

The **inferior thyroid artery** is a branch of thyrocervical trunk . It may arises directly from the subclavian artery in 15% cases.[4]

Then the artery divides into two or more branches , while crossing the recurrent laryngeal nerve .Recurrent laryngeal nerve course anteriorly or posteriorly to the artery, or in between its branches . The lowest branch supply the inferior parathyroid gland through small twig . It also supplies the lower pole of the thyroid gland.

The upper branch supplies the posterior surface of the gland, and anastamose with the descending branch of the superior thyroid artery.

The inferior thyroid artery may be absent in 2 % and 5% of individuals on the right and left side, sometimes double. [5,6]

The thyroid ima artery is a single and inconsistent artery. It originates from the brachiocephalic artery, the right common carotid artery, or the aortic arch. It occurs in about 10 percent of individuals, according to Montgomery.[7]

VENOUS DRAINAGE

The **superior thyroid vein** accompanies the superior thyroid artery. Arising from the upper pole of the thyroid, it drains into the internal jugular vein alone or with the common facial vein.

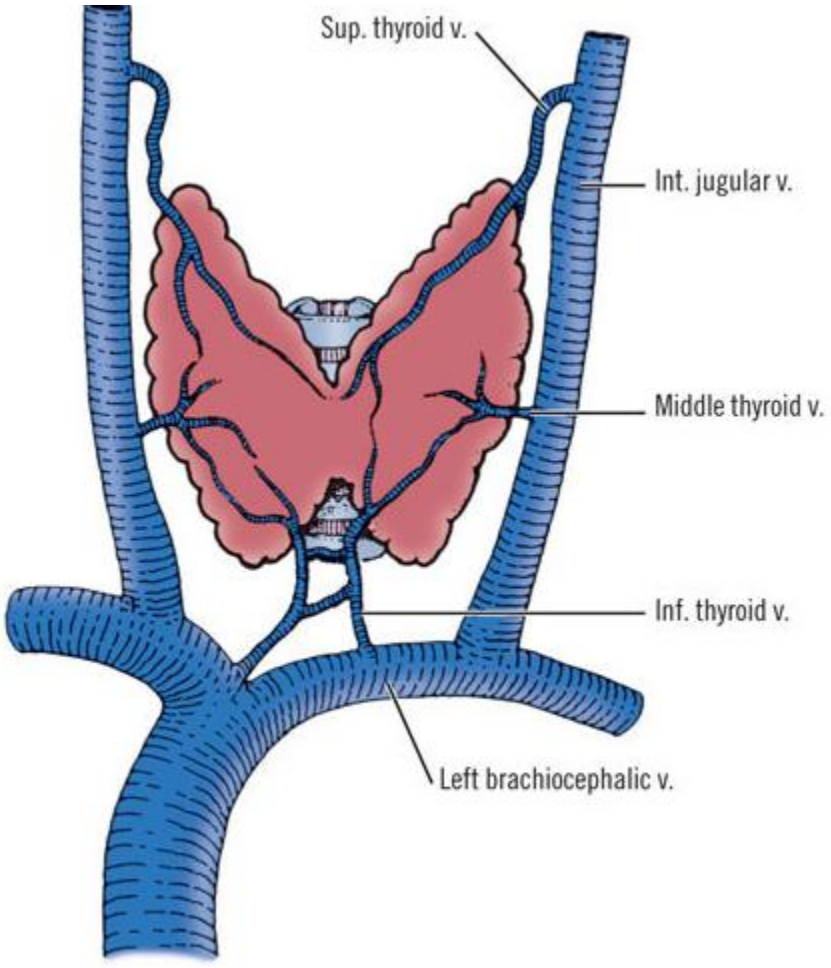


Figure 4: VENOUS DRAINAGE OF THYROID GLAND

The **middle thyroid vein** arises on the lateral surface of the gland .

It drains into the internal jugular vein. It may be absent or sometimes double and called "fourth" thyroid vein. It is vulnerable to injury during thyroid surgery.

The **inferior thyroid vein** is large and has variable course and asymmetrical on both sides. The right vein drains into right brachiocephalic vein.

The left vein drain into left brachiocephalic vein. [8]

The blood flow to thyroid gland ranges from 4 to 6 mL/min/g, excess when compared to the blood flow of the kidney (3 mL/min/g).

In diffuse toxic goiter due to Graves disease, blood flow may exceed 1 L/min and usually associated with a palpable thrill an audible bruit.

HISTOLOGY

Thyroid gland is composed of closely packed spherical units called *follicles.*They are invested within a rich capillary network. The follicular cells vary in height with the degree of glandular stimulation. It becomes columnar when active and cuboidal when inactive. The epithelium rests on a basement membrane rich in glycoproteins. It separates the follicular cells from the surrounding capillaries.

Around 20 to 40 follicles are demarcated by connective tissue septa to form a Lobule which is supplied by a single artery. The follicles of the thyroid gland are round, ovoid or tube-like hollow organs with a diameter between 0.1–0.8mm. Their walls consist of a single-layered epithelium.

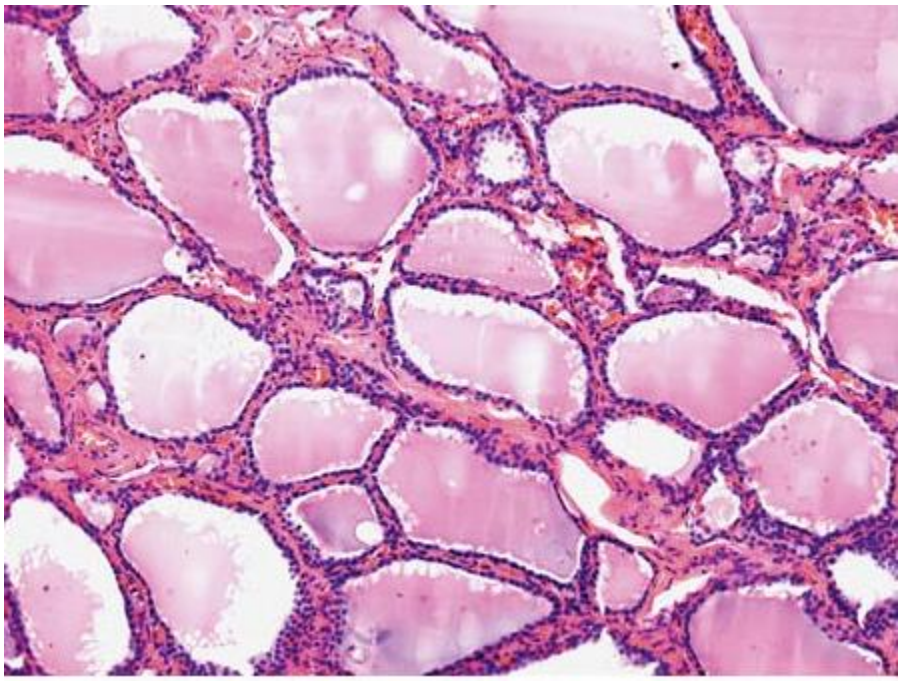


FIGURE 5 : THYROID FOLLICLE WITH COLLOID

The thyroid gland stores large amount of its hormone containing secretory product in the extracellular space. First, a liquid secretion of low viscosity is released into the follicular lumen. This secretory product is called *colloid* (mostly thyroglobulin). Thyroglobulin is the carrier for thyroid hormones.

Colloid secretion leads to the storage of thyroid hormones inside the follicle. This entrapment of thyroid hormone in thyrogloulin is called "Thyroglobulin scaffolding".[10]

Another group of thyroid secretory cells is called the " C cells or parafollicular cells". It secretes the hormone calcitonin. They are found as individual cells or clumped in small groups in the interfollicular stroma and located in the upper poles of the thyroid lobes.

PARATHYROID GLAND

HISTORICAL REVIEW

In 1849, Sir Richard Owen made the first description of the parathyroid gland after autopsy of an Indian rhinoceros. The first human parathyroids were coined by Ivar Sandstrom in 1879 ,a medical student in Uppsala, Sweden.[11]

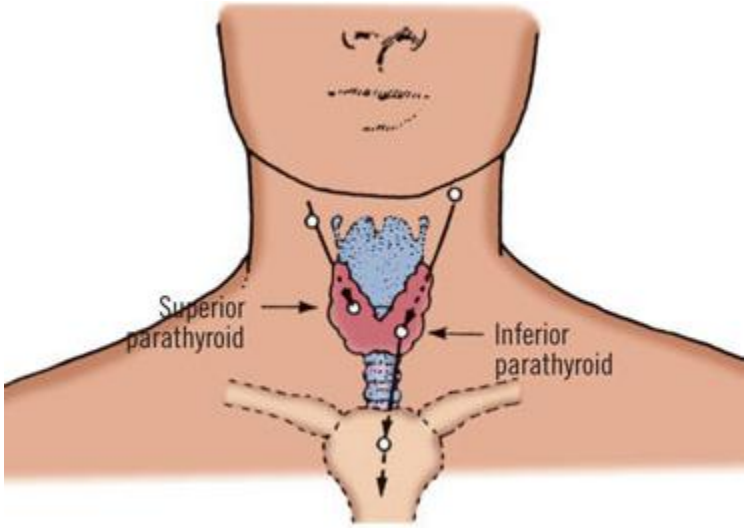


Figure 6: DESCENT OF PARATHYROID

EMBRYOLOGY

The parathyroid glands are four in number . It develop as a epithelial thickenings of the dorsal endoderm of the third and fourth branchial pouches. It subsequently migrates and as a result, the derivatives of the 3rd pouch develop into lower parathyroids and the fourth pouch develop into the upper parathyroids .The position of superior parathyroid gland is constant, usually found at the posterior surface of the superior and middle thyroid lobes at the level of cricoid cartilage .[11]It has its own capsule of connective tissue , sometimes present in the thyroid capsule, sometimes may even follow a blood vessel deep into a sulcus of the thyroid.[8]

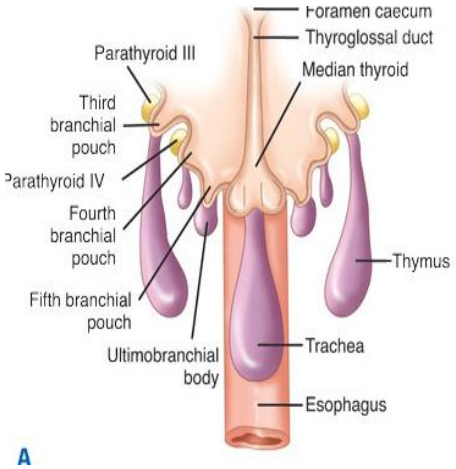


Figure 7 : PARATHYROID EMBRYOLOGY

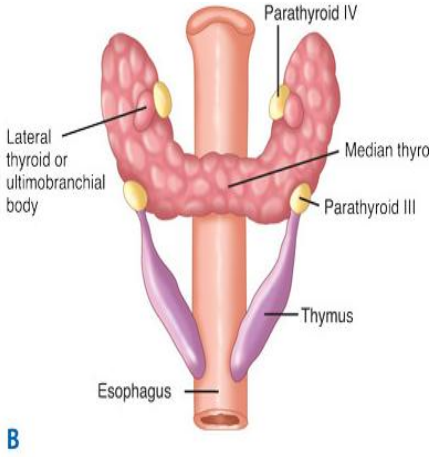


Figure 8: PARATHYROID LOCATION

RELATIONS OF PARATHYROID GLAND

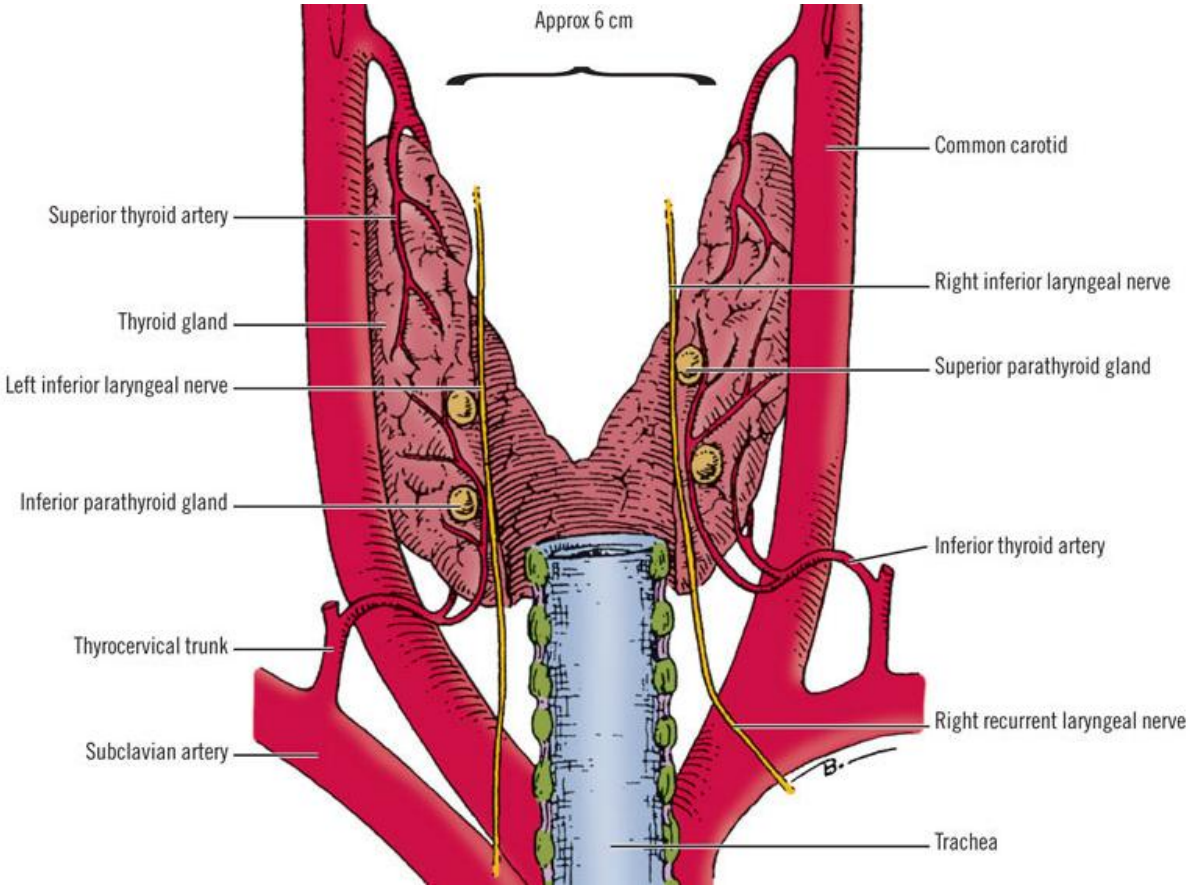


Figure 9 : PARATHYROID GLAND AND REC.LARYNGEAL NERVE

Parathyroid sometimes found at level of carotid bifurcation or mediastinum but it is rare. During surgery , one should trace from the point of entry of inferior thyroid artery into thyroid gland. The parathyroid glands usually located one inch within this point.

HISTOLOGY

The major part of the parathyroid gland parenchyma is made of chief cells and oxyphil cells. They are in trabeculae within a stroma consisting of fat cells. All the parathyroid cells , particularly chief cells secrete the parathyroid hormone and regulates metabolism of calcium and phosphate.

Oxyphil cells are acidophilic containing mitochondria , derived from chief cells.

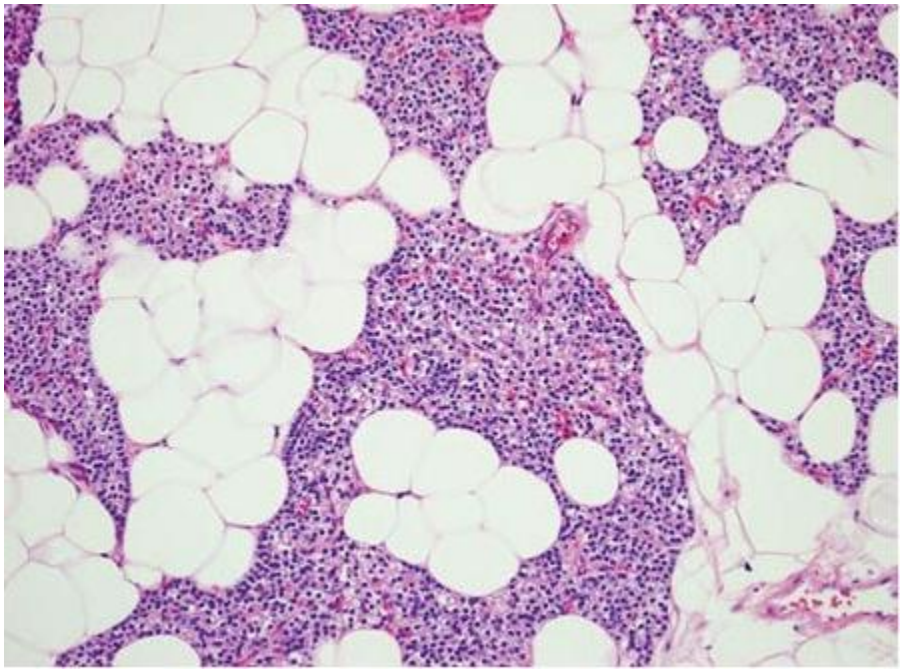
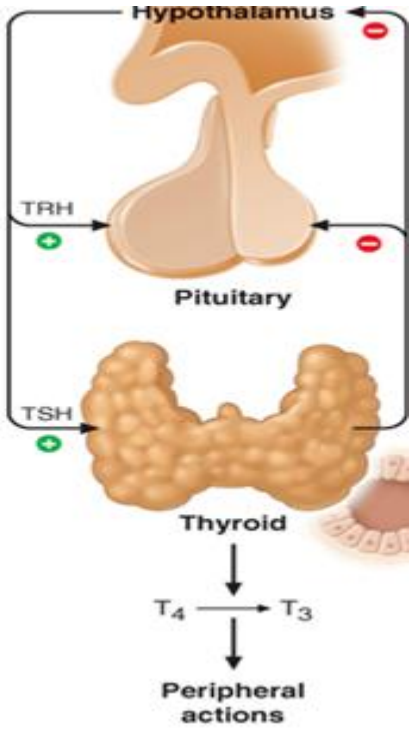


Figure 10 : PARATHYROID HISTOLOGY

THYROID HORMONE REGULATION

TSH, is secreted from thyrotrope cells situated in the anterior pituitary. It plays a key role in thyroid regulation and the physiologic marker of thyroid hormone actions. TRH , a positive regulator from hypothalamus stimulates production of TSH from anterior pituitary. [12] TSH stimulates thyroid hormone synthesis and secretion.



Thyroid hormones act predominantly through thyroid hormone beta 2 receptor. This negative feedback inhibit TRH and TSH production. TSH is released in a pulsatile fashion and exhibits a diurnal variation its highest levels occur at night.

THYROID HORMONES SYNTHESIS AND SECRETION

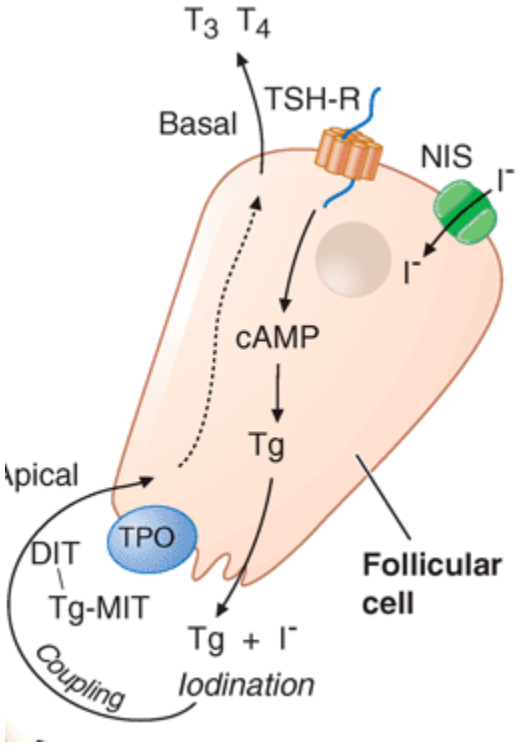
The thyroid hormones T3 and T4 are synthesized from thyroid gland by

1. Iodide trapping

active transport of iodide across the basement membrane into the thyroid cell .

2.Organification

oxidation of iodide and iodination of tyrosyl residues in thyroglobulin .



3. Coupling

pairs of iodotyrosine molecules within thyroglobulin to form the iodothyronines T3 and T4 .

4. Release of thyroid hormones

Pinocytosis and then proteolysis of thyroglobulin with release of free iodothyronines and iodotyrosines into the circulation.

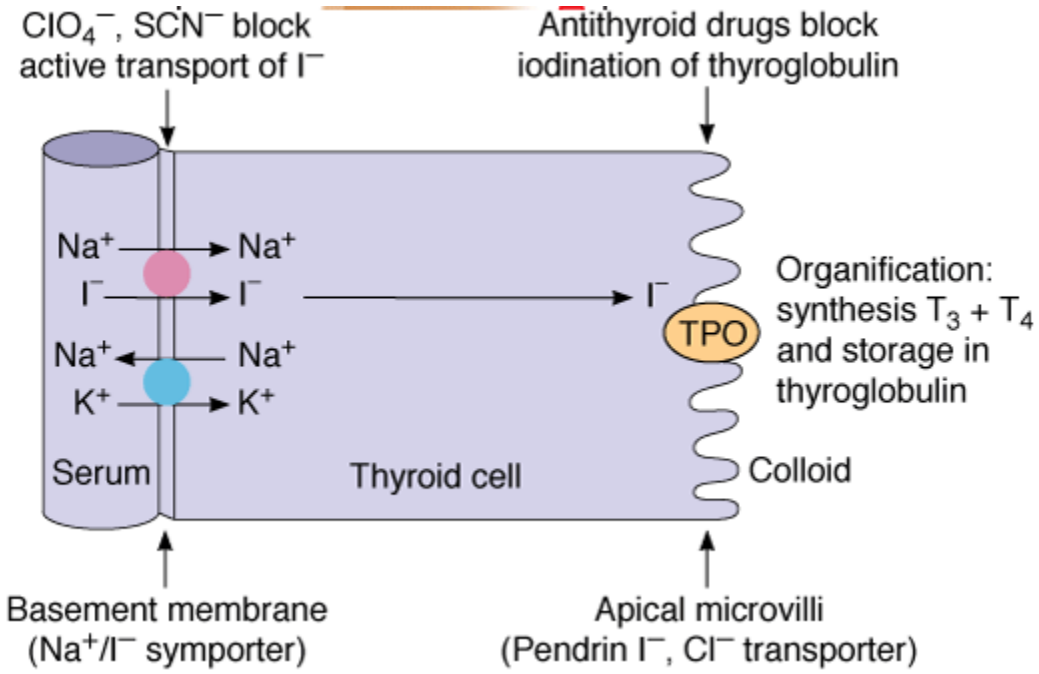
5. Deiodination of iodotyrosines within the thyroid follicle, conservation and reuse of the liberated iodide; and

6 . Intrathyroidal 5'-deiodination of T4 to T3.

IODINE

Iodine is a major structural component of thyroid hormones and it is an essential micronutrient. It is consumed in food or water as iodide. It is excreted mostly by the kidneys as iodide , measurement of urinary iodide is an excellent index of dietary intake.[13]

WHO defined dietary iodine deficiency as , a daily iodine intake less than 100 micro gram /day, When iodide intake is less than 50 microgram/day it results in Thyroid gland enlargement (goiter) and ultimately hypothyroidism.



Thyroid cells express more amount of sodium-iodide symporter (NIS), for active transport of iodide from the blood. [14]The thyroid gland uses only a fraction of the iodide supplied to it for hormone synthesis, and the rest returns to the extracellular fluid pool.

PHYSICAL EXAMINATION

Examination of the neck is best done with the patient seated in good light with the neck relaxed. The physician should first inspect the neck, especially while the patient swallows to determine whether it moves with swallowing.

The patient should be provided with a cup of water to facilitate swallowing. Movement on swallowing is a characteristic feature of the thyroid gland because it is ensheathed in the pretracheal fascia. This feature differentiate thyroid swelling from most other neck masses. The position of the trachea should be noted. Then note the shape and size of the gland and its consistency.

Diffuse goiter and the hyperplasic gland of the hyperthyroid patient with Graves' disease may be softer than normal, the gland of Hashimoto's disease is usually firm. If nodules are palpated, their number , shape, size, position, translucency, and consistency in relation to the surrounding tissue should be determined.

Finally, palpation should always include examination of the regional lymph nodes pre tracheal , prelaryngeal, nodes along the jugular vein, posterior triangle,and in the supraclavicular region. Auscultation of the neck may confirm the increased vascularity of an enlarged, hyperactive gland suggesting Graves' disease.

An arm-raising test is useful when a retrosternal goiter is suspected when congestion and venous engorgement of the face and sometimes respiratory distress. *(Pemberton's sign).*

EVALUATION OF PATIENTS WITH THYROID DISEASE

Laboratory tests :

These tests are done to

(1) assess the state of the hypothalamic-pituitary-thyroid axis.

(2) estimates the T_4 or T_3 concentrations in the serum.

(3) assess the impact of thyroid hormone on tissues.

(4) the presence of autoimmune thyroid disease.

THYROID STIMULATING HORMONE: (Normal 0.5–5 U/mL)

Serum TSH reflects the ability of the anterior pituitary to detect free T4 levels. Rate of TSH secretion is sensitive to the plasma concentrations of free thyroid hormones , hence it provides a precise and specific test of the thyroid status of the patient. There is an inverse relationship between the free hormones and the serum TSH concentration.

The free α-subunit common to TSH, FSH, LH, and hCG is commonly detectable in serum with a normal range of 1 to5μg. The ultrasensitive TSH assay is the most sensitive and specific test for diagnosing hyperthyroidism and hypothyroidism . It is also used in optimizing Thyroxine treatment.

THYROID HORMONES:

The Total T3 levels indicate peripheral thyroid hormone metabolism in the non stimulated thyroid gland. Total T4 levels reflect the output from the thyroid gland, hence they are not suitable as a routine screening test.

Increased Total T4 levels seen not only in hyperthyroid state but also in conditions like elevated thyroglobulin levels secondary to pregnancy, oral contraceptives.

Decreased Total T4 levels seen in hypothyroidism and conditions associated with decreased thyroglobulin due to use of anabolic steroids and protein losing disorders.

Free T3 is most useful to confirm the diagnosis of early hyperthyroidism, where the free T3 and T4 levels rise before total T4 and T3.

NORMAL VALUES:

Total T4 (55–150 nmol/L) and T3 (1.5–3.5nmol/L)

Free T4 12–28 pmol/L) and Free T3 (3–9 pmol/L)

THYDOID ANTIBODIES

Anti Thyroglobulin antibody and Anti Thyroperoxidase antibody levels do not determine thyroid function, but it is an indicator of Autoimmune Thyroiditis. Thyroid antibody levels elevated in 80 % of the patients with Hashimoto's Thyroiditis. It may also increased in patients with Graves' disease, multinodular goiter, and sometimes in thyroid neoplasm's.

THYROGLOBULIN

Normally Tg is not released into the circulation in large amounts, but increases enormously in destructive changes of the thyroid gland, such as thyroiditis, or overactive states such as Graves' disease and toxic multinodular goiter.

Serum Tg level is used in monitoring patients with differentiated thyroid cancers for recurrence, particularly after total thyroidectomy and RAI ablation.

Elevated anti-Tg antibodies interfere with the accuracy of serum Tg levels , hence it should always be measured when assessing Tg levels. [11]

Normal circulating TBG concentration is 64 to 142 nmol/L (5 to 11 μg/dL).

PARATHORMONE

Physiology

PTH is an 84-aminoacid single chain peptide. The amino-terminal part is highly conserved and is important for the various functions. PTH is initially synthesized as (pre-pro-parathyroid hormone,).

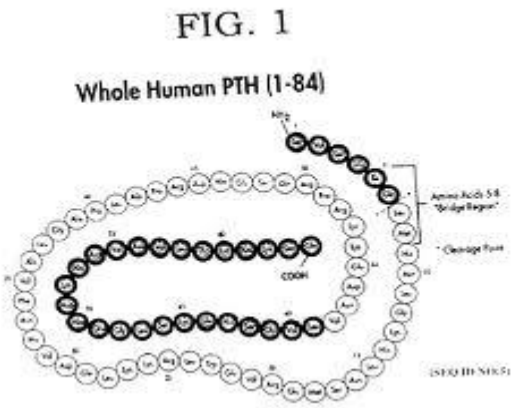


Figure 11: PARATHORMONE PEPTIDE CHAIN

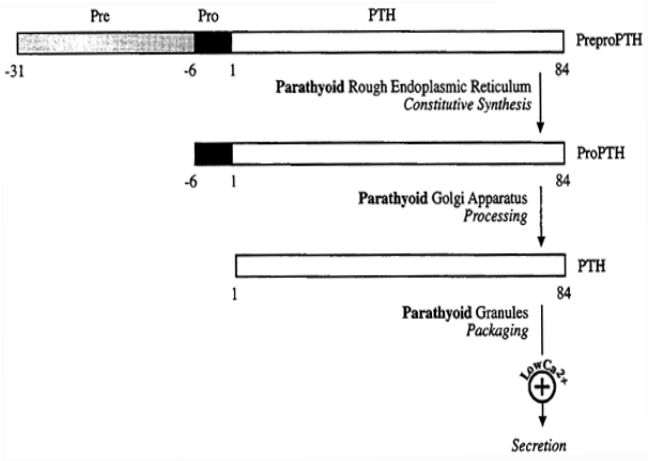
PARATHORMONE SYNTHESIS

First step:

Cleavage of amino acids . Removal of the "pre" sequence of 25 amino acid residues.

Second cleavage step:

Removes the "pro" sequence of 6 amino acid residues before secretion of the mature peptide comprising 84 residues.[12]



History of Parathormone Measurement in Humans

In 1987, Nussbaum et al. described a new method to measure the

intact (1-84) parathormone by using a two-site antibody technique which proved to be more sensitive and specific than other previous assays. They suggested that PTH had a rapid rate of decay and its half-life is 3 to 5 minutes. They later suggested that intraoperative measurement of PTH may benefit the surgeon performing parathyroidectomy.

In the assay proposed by Nussbaum et al, By heating and shaking the antibodies with the unknown sample of hormone to speed reaction times an intraoperative PTH assay (QPTH) was shown to predict postoperative calcium levels in patients with primary hyperparathyroidism.

After this assay, methods changed from radionuclear to immunochemiluminescent technology. Later it became a practical test for intraoperative measurement of PTH . Nowadays , Rapid PTH assays became commercially available for intraoperative use and used worldwide as a surgical adjunct.

PARATHYROID REGULATION

Any conditions leading hypocalcemia, due to calcium deficiency is counteracted by an increased secretion of PTH.

This in turn (1) increases the of bone resorption rate, thus releasing calcium into blood.

(2) reduces the renal excretion of calcium, increasing calcium reabsorbtion at the glomerulus filtration

(3) increases the efficiency of calcium absorption in the intestine by stimulating the production of $1,25(OH)_2D$.

Immediate action over serum calcium level is due to the effects of parathormone on the bone , lesser extent on the kidney.

As the serum calcium falls, PTH secretion raises five times to a value of about the basal rate of secretion. The ionized calcium is the important determinant of hormone secretion.

The PTH maintain the extracellular fluid (ECF) calcium concentration. It is due to acts direct action on bone and kidney and indirectly through the intestine. It increases the synthesis of $1,25(OH)_2D$ resulting in increase of

calcium. The concentration of serum ionized calcium regulates the PTH production .

PTH secretion is controlled extracellular calcium level by interaction with a calcium sensor, a G protein coupled receptor (GPCR) via Ca^{2+} ions ,the primary ligand. PTH is responsible for minute to minute regulation of plasma calcium concentration and the occurrence of hypocalcemia indicates a failure of the homeostatic action of PTH.

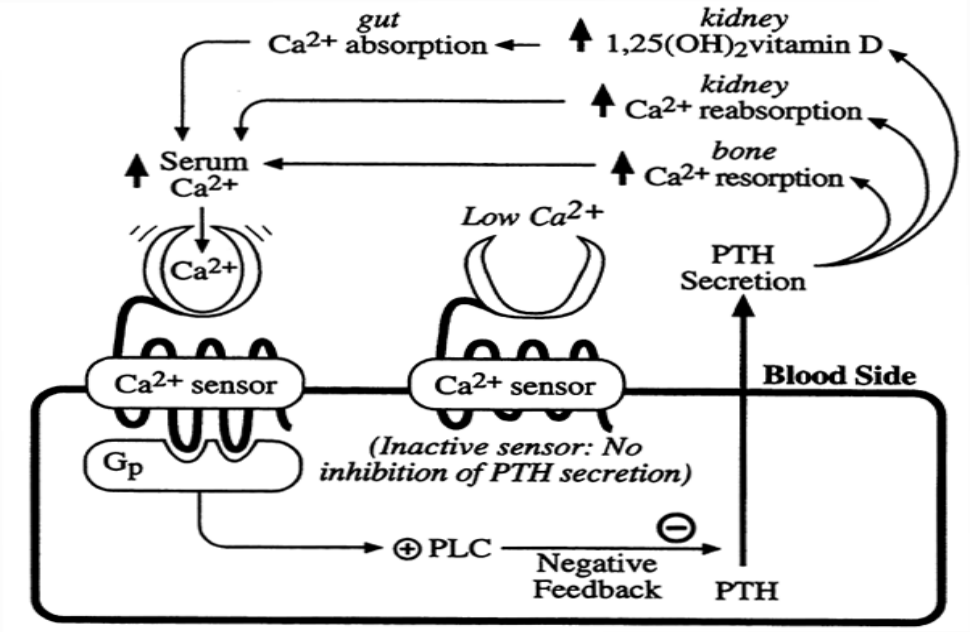


Figure 12: **Parathyroid Hormone Secretion and Calcium Homeostasis in the Parathyroid Cell**

PTH plays a major role in renal phosphate excretion. It rapidly reduces sodium phosphate transporters which is responsible renal phosphate reabsorption.

CALCIUM HOMEOSTASIS

Calcium is the one of the most common element . It plays a major role in mineralization of bones. It is an essential element available through dietary sources. Depending on age daily requirement of calcium ranges from 1000 to 1500 mg/day.[15]

Calcium other than from bones, is <1% of total body calcium The constant and rapid exchanging property of calcium is responsible for essential functions such as intracellular and extracellular signaling, nerve impulse transmission, and muscle contraction .[16,17]

Calcium homeostasis is maintained through an integrated hormonal mechanism. This system controls calcium transport in the intestine , kidney, and bone. The hormones responsible calcium regulation are PTH, $1,25(OH)_2D$, serum ionized calcium . The receptors are PTH receptor (PTHR)and and the vitamin D receptor (VDR) and the calcium-sensing receptor (CaR). [18, 19]

FUNCTIONS:

It provides mechanical stability of the bones and serves as a reservoir.

It maintains extracellular fluid (ECF) calcium concentration when needed. The concentration of ionized calcium in the ECF should be maintained within a normal range .

It plays a critical role in cellular functions like neuromuscular activity and signal transduction.

FREE CALCIUM

Intracellular cytosolic free calcium levels are ~100 nmol/L and are 10,000-fold lower than ionized calcium concentration in the blood and ECF (1.1–1.3 mmol/L).

Cytosolic calcium does not play the structural role played by extracellular calcium, but it serves a signaling function. The steep chemical gradient of calcium from outside to inside the cell promotes rapid calcium influx through various membrane calcium channels.

IONIZED CALCIUM

In blood, the normal total calcium concentration is 2.2 – 2.6 m*M* (8.5–10.5 mg/dL), of which ~50% is ionized. The remainder is bound ionically to negatively charged proteins (predominantly albumin)or loosely bound with phosphate, sulfate, citrate . Any alterations in serum protein concentrations directly affect the serum calcium level, even if the ionized calcium concentration is normal.

An algorithm to correct for protein changes adjusts the total serum calcium (in mg/dL) upward by 0.8 times the deficit in serum albumin (g/dL) or by 0.5 times the deficit in serum immunoglobulin (in g/dL).

Ionized calcium in blood directly suppresses PTH secretion by activating parathyroid calcium-sensing receptors (CaSRs) and indirectly affects PTH secretion via effects on $1,25(OH)_2D$ production. This active vitamin D metabolite inhibits PTH secretion by an poorly understood mechanism of negative feedback.

REGULATION OF CALCIUM

Intestinal absorption of calcium involves both active (transcellular) and passive (paracellular) mechanisms.

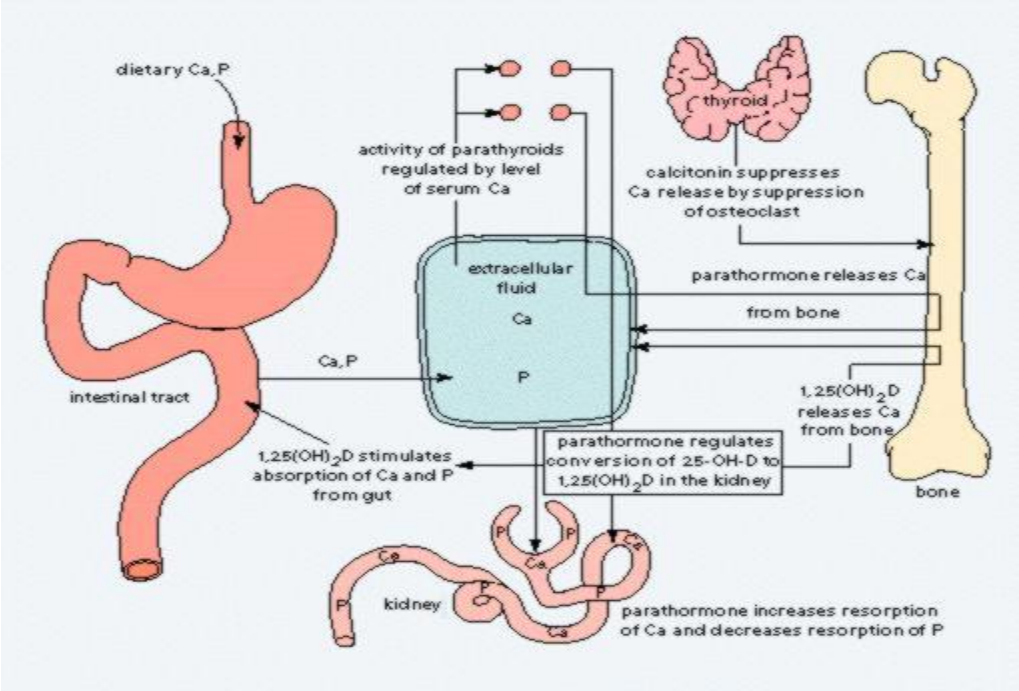


Figure 13 : CALCIUM REGULATION

Passive calcium absorption is unsaturated and approximates 5% of daily calcium intake. Active calcium absorption involves apical calcium entry via specific ion channels (TRPV5 and TRPV6), whose expression is regulated mainly by $1,25(OH)_2D$, and approximately ranges from 20 to 70% of daily calcium intake.

Active calcium transport occurs mainly in the proximal small bowel (duodenum and proximal jejunum). Optimal rates of calcium absorption require gastric acid.

The feedback-controlled hormonal regulation of intestinal absorptive efficiency results in a relatively constant, daily net calcium absorption of ~5–7.5 mmol/d (200–400 mg/d), inspite of large changes in daily dietary calcium intake.

Renal calcium Absorption

The daily load of absorbed calcium from intestine is excreted by the kidneys in a way that is tightly regulated by the concentration of ionized calcium in the blood.

Approximately 8–10 g/d of calcium is filtered by the glomeruli, of which only 2-3% appears in the urine. Most filtered calcium (65%) is reabsorbed in the proximal tubules via a passive, paracellular route that is coupled to concomitant NaCl reabsorption .

The cortical thick ascending limb of Henle's loop,(cTAL) reabsorbs roughly another 20% of filtered calcium, also via a paracellular mechanism.

PREOPERATIVE EVALUATION

Ultrasound neck:

Ultrasound is portable and excellent noninvasive imaging study of the thyroid gland. It helps in the evaluating the thyroid nodules and distinguish solid from cystic ones, and provides information about size and multicentricity. [11] Also used to assess the cervical lymphadenopathy and to guide FNAC. It has no radiation hazard, easy availability, cost effective, but has disadvantage of observer variation.

Thyroid Function Test:

Free T3, T4,TSH

Fine Needle Aspiration Cytology:

FNAC is the single most important test in the evaluation of thyroid masses. It can be performed with or without ultrasound guidance . Ultrasound guidance is usually recommended for nodules that are difficult to palpate, and for cystic or solid-cystic nodules that recur after the initial aspiration.

In FNAC, the majority of nodules are reported as benign (65%),suspicious(20%), malignant (5%),and nondiagnostic (10%).

The incidence of false-positive results is about 1% and false-negative result is3%. In case of nondiagnostic, the test should be repeated.

The risk of malignancy in benign lesion such as cysts and colloid nodule is <3% and the risk of malignancy in suspicious cytology is about 20%.

In this situation, diagnosis of malignancy relies on demonstrating capsular or vascular invasion .These features cannot be determined via FNAC

Video Laryngoscopy:

Vocal cord mobility is assessed by indirect and fiberoptic laryngoscopy.

vocal cord mobility should be documented routinely in preoperative workup and after completing surgery .

THYROIDECTOMY

Position: patient should be placed in the supine position with the arms tucked close to the side with a sandbag between the scapulae. Neck is extended to provide maximal exposure.

STEPS:

1. A Kocher transverse collar incision, typically 4 to 5 cm in length, is placed in or parallel to a natural skin crease, two finger breadths above the sternal notch .

2. skin incision has been deepened through the subcutaneous tissues and platysma , then subplatysmal flaps are raised above upto the level of the thyroid cartilage. Lower flaps raised upto the suprasternal notch.

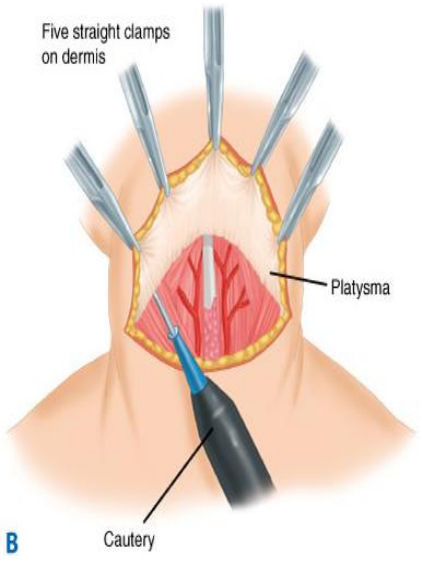


Figure 14 : RAISING SUBPLATYSMAL FLAP

3. The deep cervical fascia is incised in midline. The strap muscles are then retracted along the entire length of the mobilized flaps, and the thyroid gland is exposed .The strap muscles rarely divided to gain exposure to the thyroid gland**.**

If this is necessary, the muscles should be divided higher to preserve their innervation by branches of the ansa cervicalis.

4. Sternothyroid muscle is then dissected from the underlying thyroid by a combination of sharp and blunt dissection, to expose the middle thyroid vein.

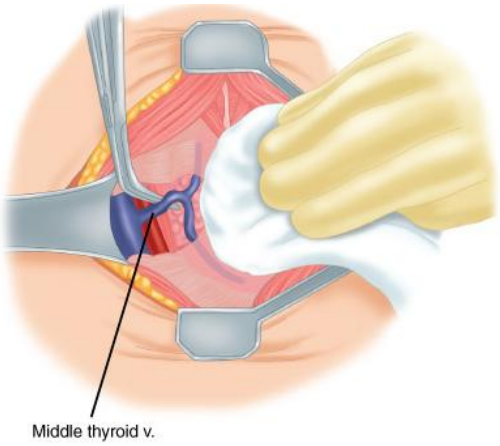


Figure 15 : MIDDLE THYROID VEIN

The thyroid lobe is retracted medially and anteriorly and the lateral tissues are swept posterolaterally using peanut sponge. Middle thyroid veins are ligated and divided.

5.The superior thyroid pole is identified by retracting the thyroid first . inferomedially and then upper pole of the thyroid gland is retracted caudally and laterally. The dissection plane is kept as close to the thyroid gland and the superior pole vessels are individually identified , ligated, and divided close to the thyroid gland to avoid injury to the external branch of the superior laryngeal nerve. Since it is not always possible to visualize external laryngeal nerve, the superior thyroid artery and vein should be separately ligated and divided.

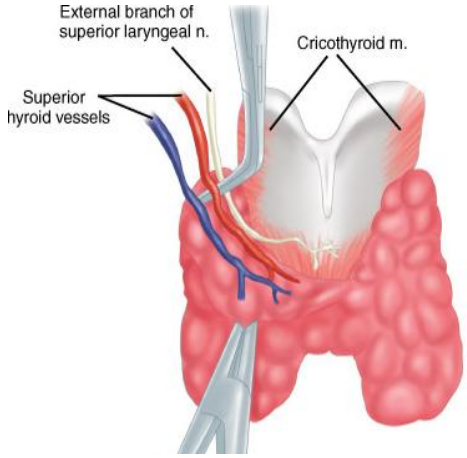


Figure 16 : LIGATION OF SUPERIOR THYROID VESSELS

5. The lower pole of the thyroid gland should be mobilized by gently sweeping. The inferior thyroid vessels are ligated individually, and divided close to the surface of the thyroid gland to minimize devascularisation of the parathyroids (extracapsular dissection) or injury to the RLN.

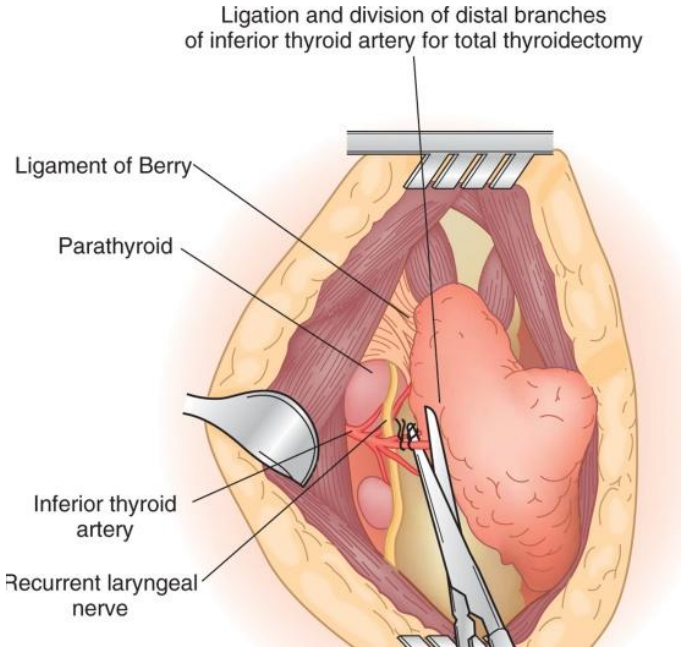


Figure 17 : LIGATION OF INFERIOR THYROID VESSEL

7.The RLN should then be identified. The course of the right RLN is more oblique than the left . The nerves can be consistently identified at the level of the cricoid cartilage. The parathyroid's usually can be identified within 1 cm of the crossing of the inferior thyroid artery and the RLN, although they also may be ectopic. The RLN is vulnerable to injury in the vicinity of the ligament of Berry. The nerve often passes through this ligament along with small crossing arterial and venous branches.

An anomalous recurrent laryngeal nerve commonly arises as a direct Laryngeal nerve and does not loop around the subclavian artery on the right or aortic arch on the left. This anomaly is more common on the right, and it results from an anomalous origin of the right subclavian artery from the descending thoracic aorta occurring in 1% of patients. If a nonrecurrent direct laryngeal nerves not identified, it is mistakenly injured during surgery.

8. Parathyroid glands situated anteriorly on the thyroid surface cannot be dissected from the thyroid with adequate vascularity. The parathyroid gland that has been accidentally removed during the thyroidectomy should be confirmed as parathyroid tissue by frozen section, divided in 1-mm fragments then reimplanted into individual pockets in the

sternocleidomastoid muscle.

The first step in identifying the lower parathyroid gland is to identify the extension of the thymus gland into the neck. This structure is called the thyrothymic ligament.

The inferior parathyroid gland is often located within or immediately adjacent to this ligament. The superior parathyroid gland is located on the posterior surface of the middle of the thyroid lobe. [9]

9.Berry ligament covers the nerve at this point. One must exercise great care in dividing this structure to unroof the nerve.

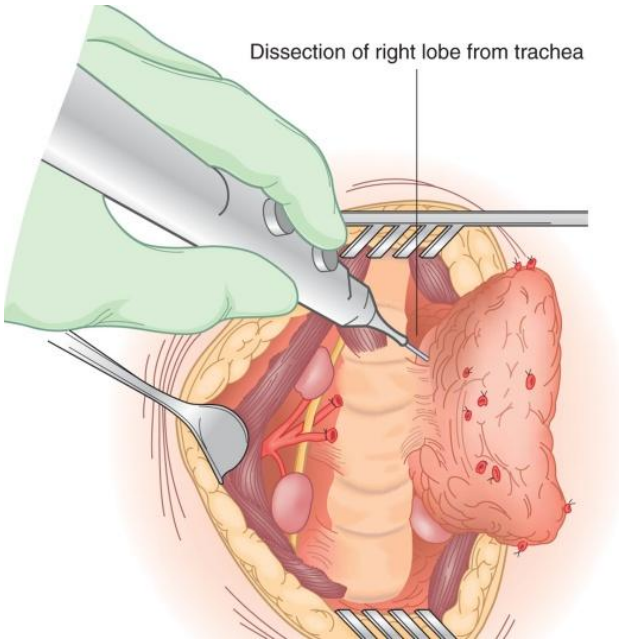


Figure 18 : DISSECTION OF THYROID GLAND FROM TRACHEA

10.The gland is then dissected from the trachea with either a knife or electrocautery.

COMPLICATIONS OF THYROIDECTOMY

MINOR COMPLICATIONS:

1.Postoperative surgical site seromas: small asymptomatic seromas resolve spontaneously or may be aspirated .

2.Poor scar formation : preventable with proper incision marking and surgical technique

3.Postoperative bleeding: Bleeding after thyroid surgery is low , incidence range(0.3-1%). If hematoma is rapidly expanding or unrecognized it can cause symptoms of airway obstruction like dyspnea, stridor.

MAJOR COMPLICATIONS:

Injury to the recurrent laryngeal nerve

Recurrent laryngeal nerve (RLN) injury results in true vocal cord palsy. Careful intraoperative identification and preservation of the RLN minimizes the risk of injury.

Injury to the Superior Laryngeal Nerve

The external branch of the superior laryngeal nerve (SLN) is commonly injured in thyroid surgery incidence around 0-25%. Preoperative counseling about his or her voice changes after surgery should be made.

Infection

postoperative infection rate following thyroidectomy is less than 1-2%. It can be prevented by sterile techniques. No role of preoperative antibiotics.

Hypothyroidism

Hypothyroidism is common following total thyroidectomy. TSH levels is the most useful investigation to detect and monitor hypothyroidism and treat accordingly.

Thyrotoxic storm

Thyrotoxic storm is a rare complication due to manipulation of the thyroid gland during surgery in Thyrotoxic patients. It can develop before, during or after surgery.

For Thyrotoxic crisis, during thyroidectomy, stop the procedure. Carefully monitor oxygenation. Administer IV beta-blockers, propylthiouracil, sodium iodine and steroids.

Post-Surgical Hypoparathyroidism and Hypocalcemia

In early 1883, Theodore Kocher described three main postoperative complications of thyroidectomy such as injury to recurrent laryngeal nerve, myxedema and tetany .

The most common causes of Hypocalcemia is Hypoparathyroidism due to

1. accidental damage or removal of the parathyroid gland

2. disruption of blood supply to the parathyroid glands during surgery.

3. “Hungry bone syndrome” due to remineralization of the bone

HYPOCALCEMIA

TYPES:

1. Transient Hypocalcemia
2. Permanent Hypocalcemia

Short-term Hypocacemia is called parathyroid gland “stunning.” If persistent more than 6 months it is called postoperative permanent Hypoparathyroidism .

The rate of transient Hypocalcemia was reported between 5-50%, is due to transient ischemia to the parathyroid glands and increased release of the acute phase reactant endothelin 1.[20]But the rate of permanent hypocalcemia 0.5-2%. Capsular dissection of thyroid lobes described in detail by Delbridge and Reeve could markedly reduce the incidences of permanent hypocalcaemia.[29]

Parathormone (PTH) was found to be useful in predicting hypocalcaemia after thyroidectomy and is recommended by Australian Society of Endocrine Surgeons. [28]

The immediate manifestations of hypocalcaemia are mostly neuro-muscular symptoms and occasionally psychosis. Ectodermal changes cause alopecia,eczema, cataract within six months of surgery. Persistent hypocalcemia may cause intracranial lesions and cardiac arrhythmias. Permanent hypocalcemia causes substantial impact on health with financial loss.

PREDISPOSING FACTORS OF POST THYROIDECTOMY HYPOCALCEMIA

1. Large goiter

2. Total thyroidectomy

3.Thyroid malignancy

4. Hyperthyroidism

5. Thyroiditis

6. Central Neck dissection

7.Recurrent goiter

8.Retrosternal extension

CAUSES OF TRANSIENT HYPOCALCEMIA

Transient hypocalcemia is seen with

1.severe sepsis,

2.burns,

3.acute renal failure,

4.extensive transfusions with citrated blood.

5.Hypoalbuminemia is the primary cause of the reduced total calcium concentration.

6.Medications such as protamine sulphate, heparin, and glucagon may cause transient hypocalcemia.

7. Patients with *acute pancreatitis* have hypocalcemia that persists during the acute inflammation.

8. Rarer causes of acquired chronic hypoparathyroidism

-include radiation injury

- hemochromatosis or hemosiderosis

Transient hypoparathyroidism is common following thyroid and parathyroid surgery. After a variable period of hypoparathyroidism, normal parathyroid function may return due to hyperplasia or recovery of remaining tissue. Occasionally, recovery occurs months after surgery.

CLINICAL MANIFESTATIONS

The hallmark of acute hypocalcemia is neuromuscular irritability. Patients usually complain of tingling and numbness over peri oral region, fingertips and toes. Paresthesia of the extremities along with fatigue and anxiety can occur. Muscle cramps can be painful leading to carpal pedal spasm or tetany.

Neuromuscular irritability:

Paraesthesia, numbness

Chvostek's sign

Trousseau's sign

Tetany

Seizures

Laryngeal spasms and Bronchospasm

Neurological signs and symptoms

Extrapyramidal signs due to calcification of basal ganglia

Personality disturbances

Irritability

Impaired intellectual ability

Parkinsonism

Choreoathetosis

Mental status:

Confusion

Psychosis

Fatigue

Anxiety

Cardiovascular manifestations:

ECG :Prolongation of the QT-interval.

Abnormal T waves.

OBJECTIVES OF THE STUDY

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1. To assess the role of Parathormone level after Total thyroidectomy in predicting the complication of hypocalcemia.

2.To correlate the serum Parathormone and corrected calcium level after Thyroidectomy.

MATERIALS AND METHODS

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SOURCE OF DATA

Data will be collected from patients admitted and underwent Total Thyroidectomy in General surgery department at Govt Kilpauk Medical College Hospital, Chennai.

PERIOD OF STUDY

From January 2013 to December 2013.

DESIGN OF STUDY

Prospective study.

SAMPLE SIZE

48 patients.

METHOD OF COLLECTION OF DATA :

Patients admitted with complaints of Thyroid swelling will be considered for this study .

1. Detailed history elicited by direct interview with the patient regarding age, sex, symptoms and thorough clinical examination.
2. Pre operative investigations like thyroid function test, Ultrasound thyroid and FNAC to confirm the diagnosis of thyroid swelling .
3. Essential investigations like serum albumin, calcium levels and calculation of corrected calcium levels.

Hyperthyroidism was controlled before surgery . Vocal cords function were assessed by indirect laryngoscopy prior to operation.

4. Post operative investigations include Intact serum Parathormone, serum albumin, calcium levels and calculation of corrected calcium levels and histopathological examination of specimen of total thyroidectomy.

Surgery was performed by members of KMC surgical team under general anesthesia. The lateral mobilization was done by the capsular dissection technique identifying parathyroid glands and laryngeal nerves .The parathyroid glands were identified and preserved in most of the cases.

Auto transplantation was done in the sternocleidomastoid muscle in few patients .Vocal cords were reassessed after extubation by direct laryngoscopy.

Hypocalcaemia diagnosed when serum corrected calcium level dropped below **8.5** mg/deciliter.

Corrected Calcium level = [0.8 X (normal albumin - patient's albumin)] + serum Calcium level.

normal albumin: 4 g/dl.

STUDY GROUP SUB CLASS

Group1: High-risk .

1. patients with postoperative symptoms of hypocalcemia (eg, tingling and numbness),

2. postoperative serum calcium levels less than 8.5 mg/dL.

3. PTH level Less than 10 pg/mL on postoperative Day 1.

Group 2: Low-risk.

All other patients in the study.

Hypothesis of this study is that, using the intact PTH level

Patients at minimal risk of hypocalcaemia*:* could be discharged early, avoiding an unnecessary extension of hospital stay.

Patients at high risk of hypocalcaemia: would initiate treatment with calcium supplements before the onset of symptoms, thus preventing the development of a potentially deadly condition.

INCLUSION CRITERIA

1.Age between 20 years to 60 years

2.Multinodular goitre

3. Toxic nodular goitre

4.Thyroid malignancies

5. Patients willing to give consent for study

EXCLUSION CRITERIA

1. Hemithyroidectomy
2. Solitary nodular goitre
3. Age <20yrs and >70yrs
4. Preoperative parathyroid gland abnormality on USG.
5. Drugs(Bisphosphonates, loop diuretics, anticonvulsant)
6. Systemic diseases

Diabetic nephropathy, Chronic Renal Failure, Hypoalbuminemia

Osteoporosis, Osteomalacia

7. Neck irradiation

PROFORMA

Patient name :

Age: Sex: M / F

IP No: Hospital:

Chief complaints:

Swelling over the neck ☐ difficulty in swallowing ☐

change in voice ☐ symptoms suggestive of hypothyroidism ☐

symptoms suggestive of hyperthyroidism ☐

Past history:

Any history of irradiation in the neck. ☐

Any previous neck surgery. ☐

Personal history: Goitrogenous foods ☐ iodine deficiency ☐

Menstrual history: menorrhagia ☐ hypomenorrhea ☐

Family history: thyroid disease ☐ malignancy ☐

Drug history: antithyroid drugs ☐ thyroxine ☐

GENERAL EXAMINATION

Vitals : Pulse rate -

Blood pressure- Temperature -

Examination of Thyroid region and Neck :

Cardiovascular system:

Respiratory system :

Central Nervous system :

Abdomen:

INVESTIGATION :

Thyroid Function Test :

Ultrasound Neck :

FNAC of Thyroid swelling :

Serum albumin :

Serum calcium:

Indirect Laryngoscopy :

ECG :

X Ray Neck:

POST OPERATIVE PERIOD

Serum Parathormone:

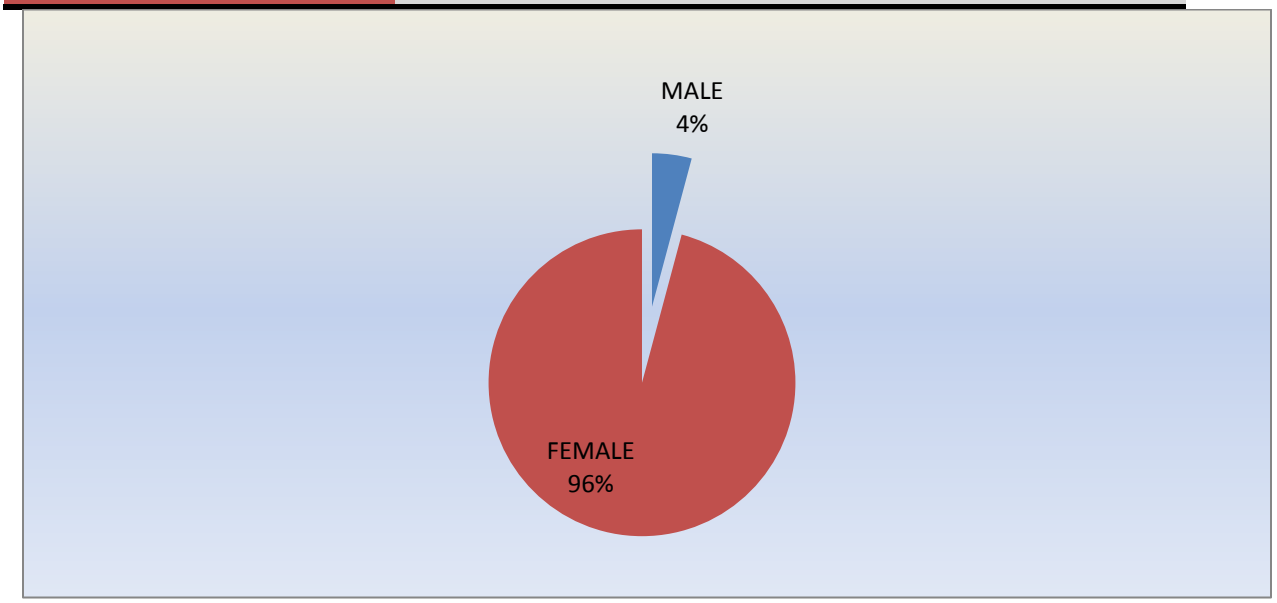
Serum calcium :

OBSERVATION AND RESULTS

SEX INCIDENCE

- About 48 patients were included in the study, 96% were female and 4% were male.
- This table shows that Thyroid disease is more common in females.

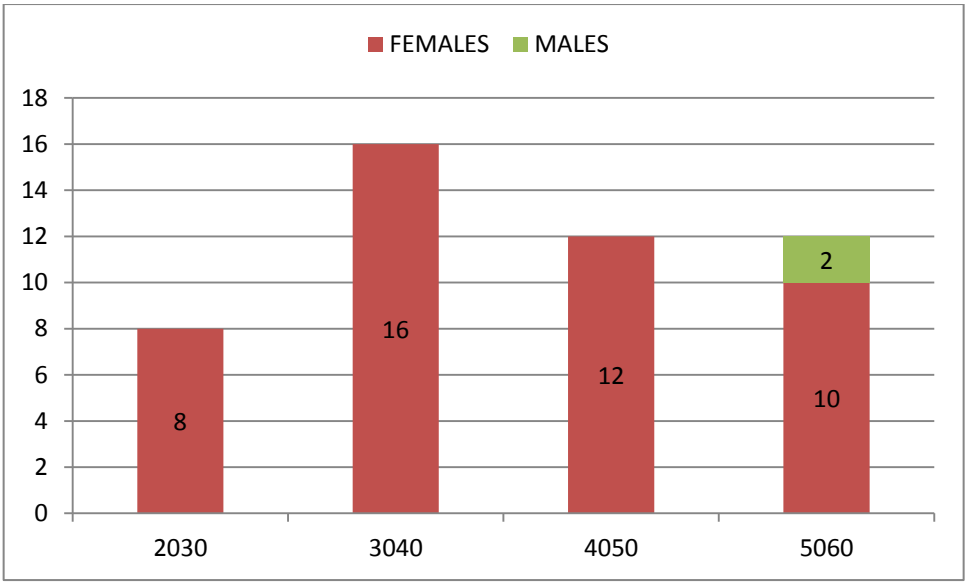
	NO. OF CASES	PERCENTAGE
FEMALE	46	96%
MALE	2	4%
TOTAL	48	100%



AGE INCIDENCE

- Incidence of female is more when compared to male and maximum at age group of 30 – 40 yrs showing the higher incidence of thyroid diseases in middle age group.

Age Group in years	Male	Female	Male %	Female %
20 - 30	-	**8**	-	**16.6%**
30 - 40	-	**16**	-	**33.4%**
40 - 50	-	**12**	-	**25.0%**
50 - 60	**2**	**10**	**4.2%**	**20.8%**

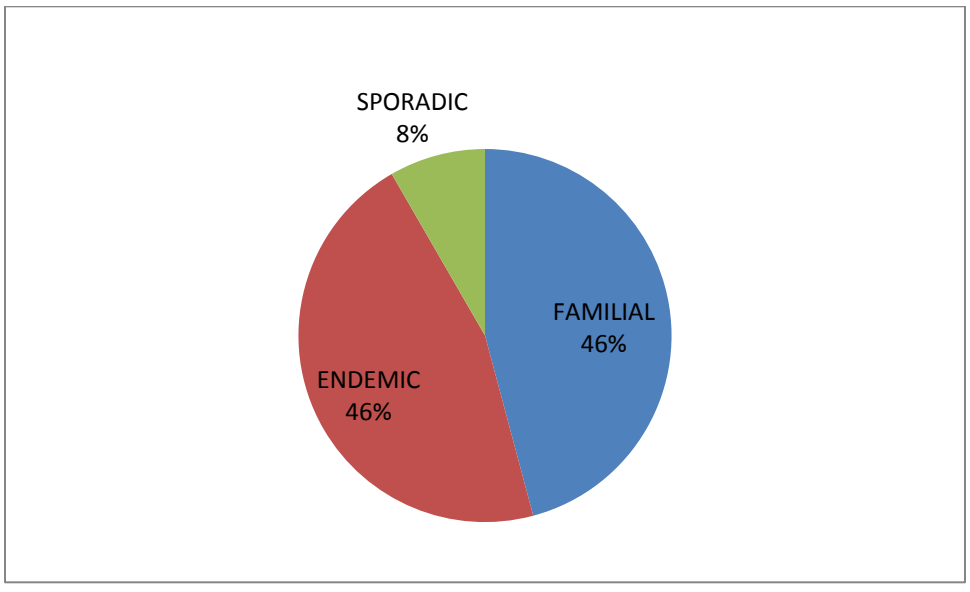


PREVALENCE :

- Out of 48 patients, 46% of patients had familial thyroid disease and 46% had Endemic goitre, 8% of them had sporadic thyroid disease.

	No. of patients	Percentage
FAMILIAL	**22**	**46%**
ENDEMIC	**22**	**46%**
SPORADIC	**4**	**8%**

- This diagram represents both endemic and familial goiters equally affect the population.



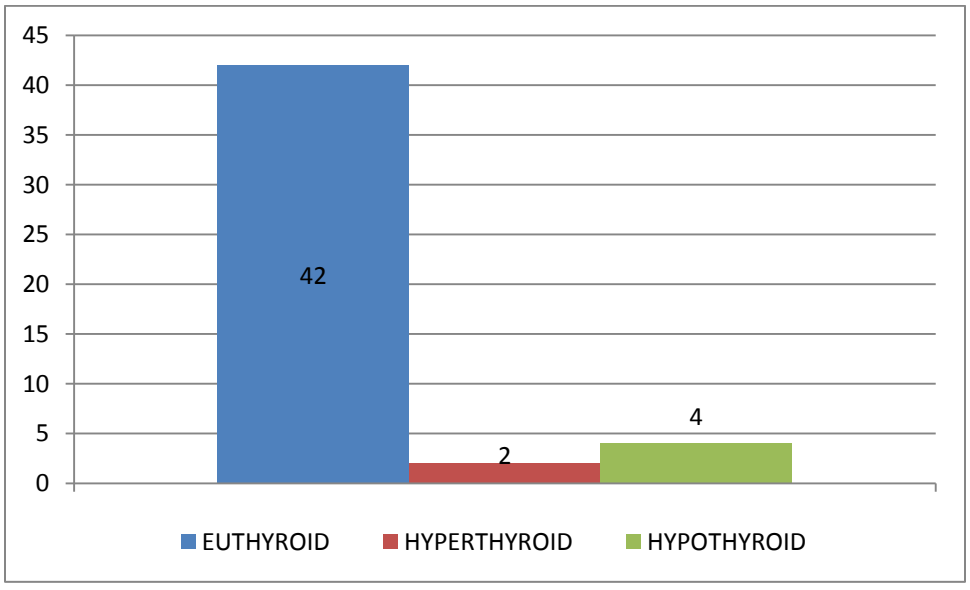
PREOPERATIVE THYROID FUNCTION TEST

	No. of patients	Percentage
EUTHYROID	**42**	**87.5%**
HYPERTHYROID	**2**	**4%**
HYPOTHYROID	**4**	**8.5%**

This table shows 87.5% were euthyroid , 8.5% patients were hypothyroid and

Only 4% patients were hyperthyroid.

- Hyperthyroid patients were started on ant thyroid drugs and posted for surgery only after attaining euthyroid state.
- Hypothyroid patients were already on thyroxine medication.

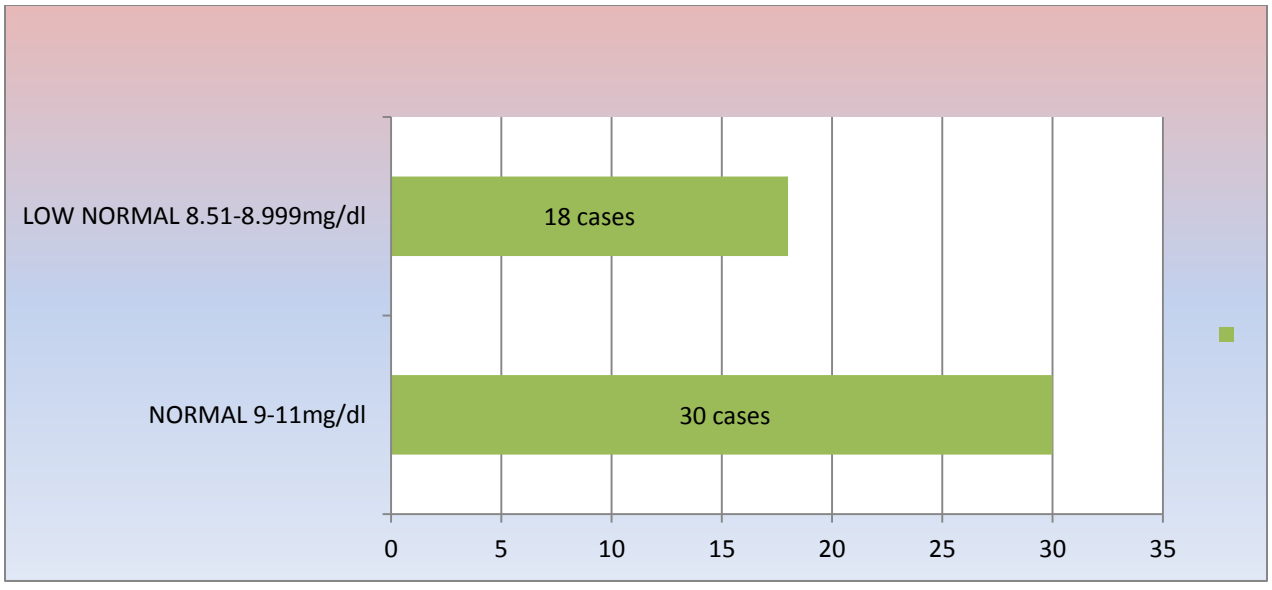


PREOPERATIVE CALCIUM LEVEL

- In 48 patients , 62.5% had corrected calcium level more than 9 mg /dl. Only 37.5% patients had corrected calcium level between 8.51 – 8.99 mg/dl.

CORRECTED CALCIUM	No. of patients	Percentage
Normocalcemia (9-11mg/dl)	**30**	**62.5%**
Mild Hypocalcemia (8.51- 8.99 mg/dl)	**18**	**37.5%**

- This chart shows , 30 cases had low risk and 18 cases had high risk out of 48 patients.



ULTRASONOGRAPHY OF THYROID SWELLING

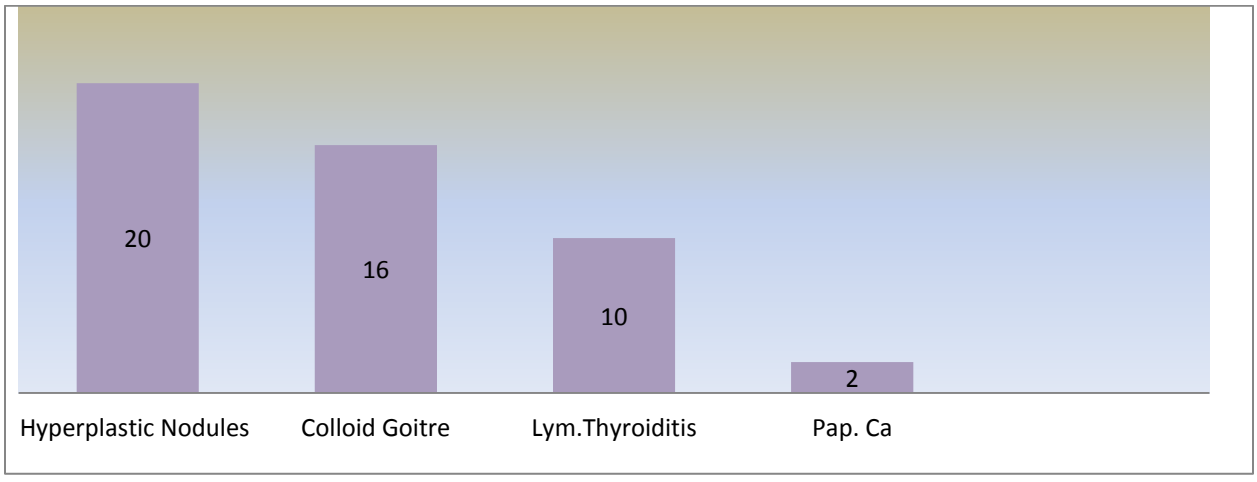
Ultrasound of the thyroid gland shows diffuse enlargement of the thyroid gland with altered echo texture in 58.3% of patients. Nodular Thyroid gland in 33.4% and complex cystic thyromegaly with without degeneration in 8.3%.

	No. of patients	Percentage
DIFFUSE THYROMEGALY	**28**	**58.3%**
MULTINODULAR GOITRE	**16**	**33.4%**
COMPLEX CYSTIC THYROMEGALY	**4**	**8.3%**

FINE NEEDLE ASPIRATION CYTOLOGY

	No. of patients	Percentage
HYPERPLASTIC NODULES	**20**	**42%**
COLLOID GOITRE	**16**	**33%**
LYMPHOCYTIC THYROIDITIS	**10**	**21%**
PAPILLARY CA.	**2**	**4%**

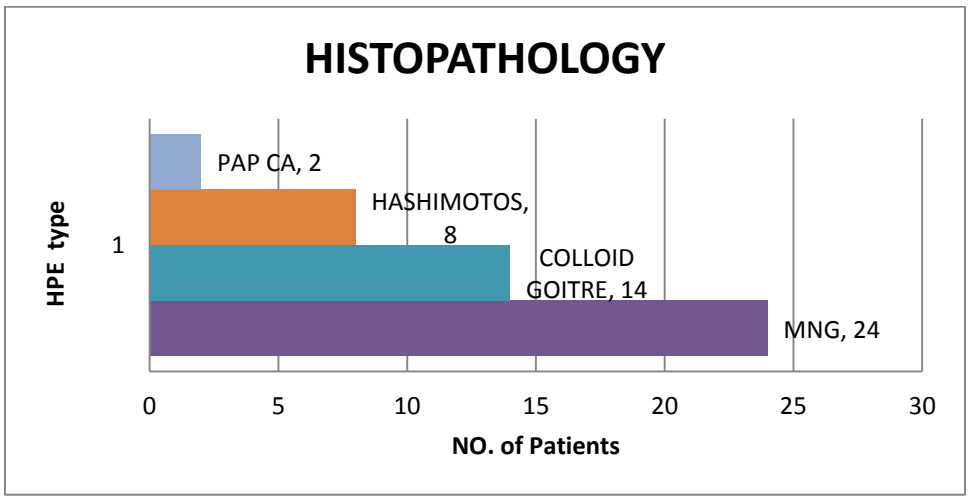
In our patients, 42% of cases FNAC reported as Hyperplastic nodules, 33% reported as Colloid goitre, 21% reported as Lymphocytic thyroiditis and only 4% reported as thyroid malignancy.



POST OPERATIVE HISTOPATHOPATHOLOGY

	No. of patients	Percentage
MULTINODULAR GOITRE	**24**	**50%**
COLLOID GOITRE	**14**	**29%**
HASHIMOTO'S THYROIDITIS	**8**	**17%**
PAPILLARY CA.	**2**	**4%**

Out of 48 patients, HPE reported as Multinodular goitre in 50% of cases, Colloid goitre in 29% of cases, Lymphocytic thyroiditis in 17% of cases and 4% reported as papillary thyroid cancer.



DATA ANALYSIS

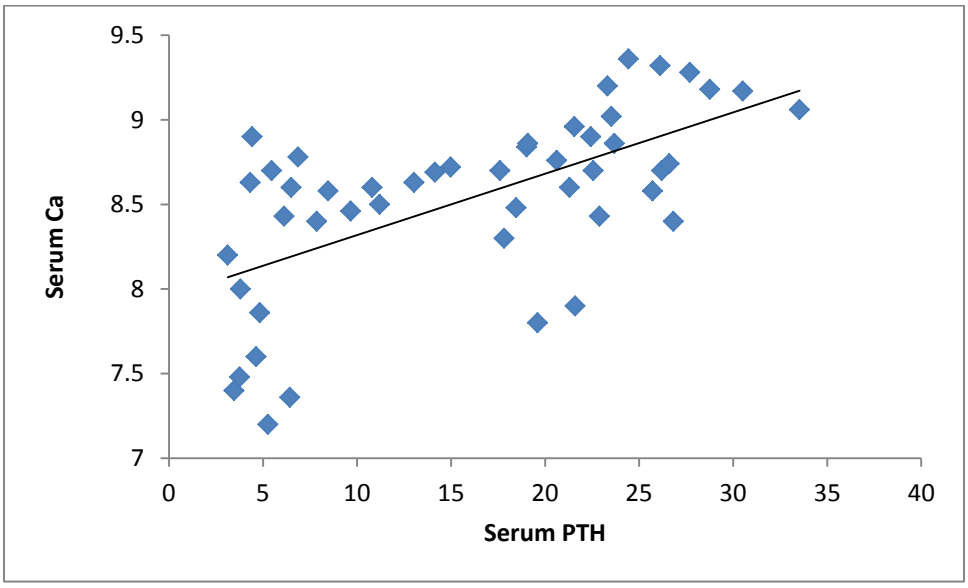
COMPARISON BETWEEN PRE OP AND POST OP CALCIUM

Parameter	Pre op	Post op	Paired t test	P value
Serum Calcium (Mean ± SEM)	9.18 ± 0.05	8.54 ± 0.07	6.816	**0.001**

SEM – standard error of mean

In this study**,** **Low preoperative serum calcium significantly correlate with fall in post operative serum calcium**. Paired t test shows the value as 6.816. **p value (0.001) is statistically highly significant.**

COMPARISON BETWEEN PTH AND CALCIUM POSTOPERATIVELY



Correlation coefficient – 0.621, P – 0.001

In this study , there is a **positive correlation between serum PTH and serum calcium done post operatively**. The Correlation coefficient (R) is 0.621. **p value (0.001) is statistically highly significant.**

COMPARING AGE GROUP WITH POST OPERATIVE HYPOCALCEMIA

Age category (years)	Risk category		Chi square	P value
	High risk	Low risk		
20-30	4	4		
30-40	12	4	6.93	0.07
40-50	3	9		
50-60	6	6		

This table shows maximum number of cases 33.4% belong to age group between 30-40yrs. Out of these 75% fall into the high risk category. Chi square value is 6.93.

Though **middle age group belongs to high risk category** , post operative hypocalcemia is not significant. **P value is not significant**.

COMPARING THYROID FUNCTION TEST WITH POST OPERATIVE HYPOCALCEMIA

Pre op TFT	Risk category		Chi square	P value
	High risk	Low risk		
Normal	23	19		
Hypothyroid	0	4	6.31	**0.04**
Hyperthyroid	2	0		

This table shows maximum number of cases seen in Euthyroid state but Statistically not significant. **Hypothyroid patients categorized only in low risk group** and the **p value is statistically significant p (0.04)** .

COMPARISON OF USG WITH POST OPERATIVE HYPOCALCEMIA

USG	Risk category		Chi square	P value
	High risk	Low risk		
Diffuse Thyromegaly	11	17		
MNG	13	3	8.47	**0.01**
Complex	1	3		

This table shows maximum number of cases about 58.3% diagnosed as Diffuse goitre and the p value is not significant. **Multinodular goitre represents 16 cases, out of these 81% fall into high risk group. P value is significant. p(0.01)**.

COMPARISON OF FNAC AND POST OPERATIVE HPE WITH POST OPERATIVE HYPOCALCEMIA

FNAC finding	Risk category		Chi square	P value
	High risk	Low risk		
Hyperplastic Nodules	15	5		
Colloid goitre	6	10	11.54	0.009
Lymphocytic Thyroiditis	2	8		
Papillary Ca	2	0		

HPE finding	Risk category		Chi square	P value
	High risk	Low risk		
Multi nodular goitre	17	7		
Colloid goitre	4	10	10.67	0.01
Lymphocytic thyroiditis	2	6		
Papillary Ca	2	0		

From the above results, **FNA biopsy** featured as colloid goitre in 33.3% and 29% in **post operative HPE** . Both the values are **significantly correlate with low risk category**. P value is statistically significant. p (0.01).

DISCUSSION

Our Institutional statistics showed that , **95.8% of thyroid operations** are for **benign diseases** which encompass more number of nodular goiters, colloid goitre and less cases of hypothyroidism, and nodules with suspicious cytology.

This study estimated **the incidence of hypocalcemia after total thyroidectomy** was **47%** compared to the incidence of hypocalcemia in the nationwide inpatient sample study which was found to be 9% only.[20] In our study, we excluded the hemithyoidectomy patients , hence the incidence was higher. Hypocalcemia significantly occurs often after total thyroidectomy than after hemithyroidectomy or lobectomy. [21]

Patients who underwent thyroidectomy with concomitant **neck dissection** were prone to develop hypocalcemia when compared to total thyroidectomy alone.[22]

Inferior parathyroid glands are at risk of injury or removal or ischemia during clearance of para-tracheal and pre-tracheal nodes in the central neck.[30]

In our study, 2 cases diagnosed as papillary ca thyroid and proceeded with total thyroidectomy with neck dissection, both patients developed hypocalcemia post operatively.

We categorized, one third of patients in the **age between 30-40yrs** as high risk group , with no significant risk of hypocalcemia among younger patients excluding malignancy. In NIS, revealed statistically increased risk of hypocalcemia for patients less than 45 years compared to older age group.[23]

Some studies reported as Hypocalcaemia is relatively more common following thyroidectomy in **hyperthyroidism**, which was observed by Miche and colleagues in 1965.[31]

We analyzed that hypothyroid patients are less likely associated with post operative hypocalcemia . Only 4% of hyperthyroid patients were operated in our study and they developed hypocalcemia.

The cause of hypocalcaemia after thyroidectomy in Grave's Disease is not known. It may be due to loss of bone mineral density.[32] Transient hypocalcaemia may be related to osteodystrophy seen in hyperthyroidism.

Our study estimated the correlation between the preoperative serum calcium with post operative Hypocalcemia. Any **low level in preop serum calcium level was significantly associated with hypocalcemia.**

The incidence of hypocalcaemia was higher in Nontoxic Nodular goiters due to accidental removal of the parathyroid gland was significantly associated with hypocalcaemia .[32,33]

This study shows that, 50% of the cases were reported as **Multinodular goite** .

Within this group, 70% cases were high risk category associated with hypocalcemia.

According to Thomusch *et al*., lateral ligation of inferior thyroid artery is strongly associated with hypocalcaemia. The capsular dissection method ensures adequate vascularity of the parathyroid glands .[34]

It was noticed in our study that inadvertent injury to vessel supplying the parathyroid or handling of the parathyroid gland cause hypocalcemia which may be due to ischemia .

Our study correlated the serum parathormone with calcium level immediately after total thyroidectomy and estimated the role of PTH to identify hypocalcemia. Out of 48 patients, 47% developed hypocalcemic symptoms. Of these symptomatic patients , 69% of them were grouped as high risk category. There is a **significant relationship between post operative PTH** and hypoparathyroidism induced **hypocalcemia**. These patients were administered calcium supplementation .

Lombardi et al. states that value of parathyroid hormone level less than 10 pg/ml after 4 hrs of surgery were unable to accurately predict hypocalcemia in 13.4% of their study population, out of 2.1% symptomatic patients. [24] .

McLeod et al. reported that postoperative PTH levels less than 12 pg/ml was 100% sensitivity and 92% specificity for predicting hypocalcemia.[25]

This study was correlated with the work of Sywak et al. who demonstrated a sensitivity of 90% and a specificity of 84% of PTH levels from 3–10 pg/ml drawn 4 hrs after surgery for predicting postoperative

hypocalcemia (which was defined by corrected calcium levels ≤2.0 mmol/l).[26,27]

In our study, Patients categorized as high risk group, whose PTH is less than 10pg/ml were closely monitored and administer with calcium supplementation .

The patients who developed hypocalcemia had postoperative PTH level of less than 15 pg/mL after 6hrs of total thyroidectomy . These results are similar with those of other author. This study also suggested that patients with 6hrs postoperative PTH level less than 10 pg/mL should undergo intensive inpatient postoperative monitoring to identify hypocalcemia and administer with calcium supplementation.

Administration of calcium and calcitriol in such high risk patients stabilize the postoperative calcium levels, reducing the complication rate and hospital stay.

Patients undergoing total thyroidectomy postoperative PTH levels greater than 15 pg/mL after 6hrs are at extremely low risk for developing postoperative hypocalcemia and can be considered for early discharge from the hospital if they are otherwise stable.

The routine measurement of serum calcium on the next day following surgery is an additional safety investigation to identify the patients with hypoparathyroidism even among those not correctly identified by the postoperative PTH level.

LIMITATIONS

The major limitation of postoperative PTH measurement is the possibility of these values may be spuriously normal or high. It can subsequently lead to unanticipated critically low calcium levels.

CONCLUSION

Hypocalcemia following total thyroidectomy remains a significant cause of morbidity and increases the cost of procedure due to prolonged hospital stay.

The use of serum PTH postoperatively 6hrs following total thyroidectomy significantly predict the complication of hypocalcemia, thereby administering calcium supplementation earlier even before the patient develop symptoms. Thus reducing the morbidity and prolonged hospital stay.

The post thyroidectomy permanent hypoparathyroidism also depends on the type of surgery , histological diagnosis and hyperthyroidism.
Proper identification of the parathyroid gland and preserving the gland with adequate vascularity significantly reduce the complication.

MASTER CHART

S	nSexm-1f-2	Age 20-30-1 30-40-2 40-50-3 50-60-4	Prevalence Familial-1 Endemic 2 Sporadic3	Preop calciu m	PrepTFTN-1 Hypo-2 Hyper-3	USG Diff.tly-1 MNG-2 COMPLEX- 3	FNAC Nod-1 Colloid-2 Lym Titis-3 Pap.ca-4	HPE Nodule-1 Colloid-2 Lym.throiditis-3 Papca-4	POST OP PTH N-1 10-15 -2 <10 -3	POST OP CALCIUM. N -1 Mild-2 Mod-3	POST OP PTH Pg/ml	POST OP CALCIUM Mg/dl	PTH RISK LOW 1 HIGH 2	CALCIUM RISK LOW I HIGH 2
1	2	2	1	9.02	1	1	2	1	2	2	14.15	8.69	1	1
2	2	4	2	8.60	3	2	1	1	3	3	4.63	7.6	2	2
3	2	4	2	9.68	1	1	2	2	1	1	26.20	8.7	1	1
4	2	1	1	9.43	1	2	2	2	1	1	23.52	9.02	1	1
5	2	2	1	9.64	1	2	1	1	3	3	3.12	8.2	2	2
6	2	2	1	8.90	1	2	1	1	3	3	3.80	8.0	2	2
7	2	3	2	9.6	2	1	3	3	1	1	19.02	8.84	1	1

MASTER CHART

				1										
8	2	1	1	9.38	1	1	3	2	1	1	23.32	9.2	1	1
9	2	3	1	9.60	1	1	2	2	3	3	4.42	8.9	2	1
10	2	2	2	8.68	1	2	2	2	1	2	26.82	8.4	1	2
11	2	2	2	8.73	1	2	1	1	1	2	19.60	7.8	1	2
12	2	2	2	9.70	1	1	1	1	2	2	17.82	8.3	1	2
13	1	4	1	8.98	1	1	1	1	3	3	7.86	8.4	2	2
14	2	3	2	9.30	2	3	1	1	1	1	22.43	8.9	1	1
15	2	3	1	9.63	1	1	3	3	1	1	27.70	9.28	1	1
16	2	2	2	9.53	1	1	2	2	1	1	21.30	8.6	1	1
17	2	2	1	9.10	1	1	4	4	3	3	3.46	7.4	2	2
18	2	4	2	8.80	1	2	1	1	3	3	5.26	7.2	2	2
19	2	4	3	9.30	1	1	2	2	1	1	30.52	9.17	1	1
20	2	4	3	9.10	1	1	3	3	1	1	22.56	8.7	1	1
21	2	1	1	9.20	1	2	2	1	2	3	6.5	8.6	2	1
22	2	3	2	8.80	1	1	1	1	1	1	17.6	8.7	1	1
23	2	3	2	8.60	1	3	1	1	3	3	11.20	8.5	1	2
2	2	1	1	8.9	1	1	3	3	3	3	10.80	8.6	2	1

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4				0										
25	2	2	1	9.08	1	1	2	1	2	2	14.98	8.72	1	1
26	2	4	2	8.68	3	2	1	1	3	3	4.83	7.86	2	2
27	2	4	2	9.62	1	1	2	2	1	1	26.60	8.74	1	1
28	2	1	1	9.63	1	2	2	2	1	1	26.12	9.32	1	1
29	2	2	1	9.34	1	2	1	1	3	3	6.12	8.43	2	2
30	2	2	1	8.82	1	2	1	1	3	3	4.32	8.63	2	1
31	2	3	2	9.81	2	1	3	3	1	1	20.62	8.76	1	1
32	2	1	1	9.68	1	1	3	2	1	1	24.43	9.36	1	1
33	2	3	1	9.46	1	1	2	2	3	3	5.46	8.70	2	1
34	2	2	2	8.58	1	2	2	2	1	2	25.72	8.58	1	1
35	2	2	2	8.83	1	2	1	1	1	2	21.60	7.9	1	2
36	2	2	2	9.86	1	1	1	1	2	2	18.46	8.48	1	2
37	1	4	1	8.68	1	1	1	1	3	3	8.46	8.58	2	1
38	2	3	2	9.43	2	3	1	1	1	1	21.56	8.96	1	1
39	2	3	1	9.56	1	1	3	3	1	1	28.76	9.18	1	1
40	2	2	2	9.24	1	1	2	2	1	1	22.89	8.43	1	2
4	2	2	1	9.1	1	1	4	4	3	3	3.76	7.48	2	2

MASTER CHART

1				8										
42	2	4	2	8.68	1	2	1	1	3	3	6.43	7.36	2	2
43	2	4	3	9.46	1	1	2	2	1	1	33.53	9.06	1	1
44	2	4	3	9.08	1	1	3	3	1	1	23.68	8.86	1	1
45	2	1	1	9.28	1	2	2	1	2	3	6.86	8.78	2	1
46	2	3	2	8.68	1	1	1	1	1	1	19.08	8.86	1	1
47	2	3	2	8.79	1	3	1	1	3	3	13.03	8.63	1	1
48	2	1	1	8.98	1	1	3	3	3	3	9.66	8.46	2	2

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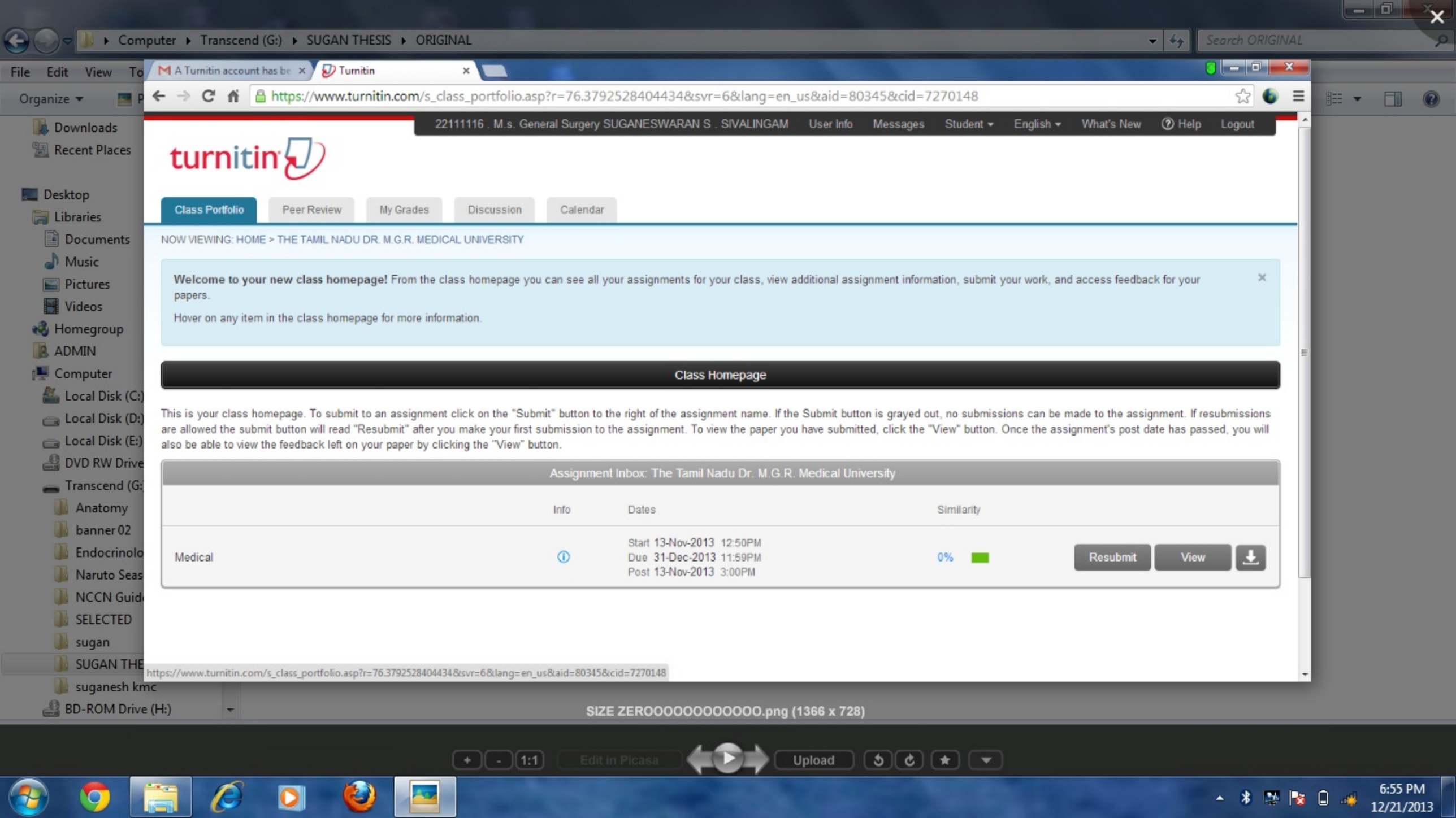
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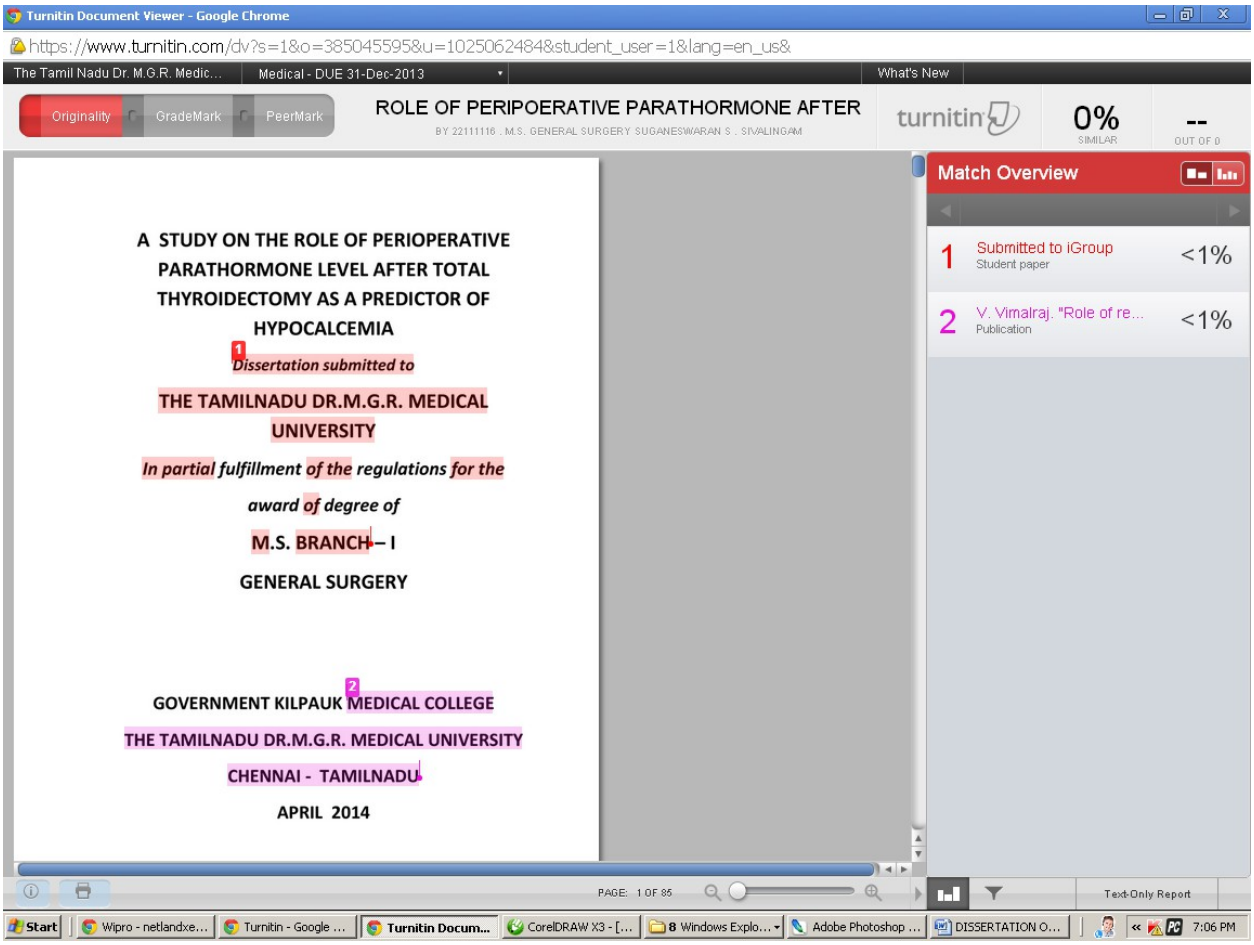
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